CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH VENTRICULAR TACHYCARDIA

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DECLARATION BY THE CANDIDATE

I hereby declare that the thesis entitled "**CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH VENTRICULAR TACHYCARDIA"** submitted towards partial fulfillment of the requirements for the award of **DM in Cardiology** degree to BFUHS is a compilation of original work carried out by me under the supervision of **Dr. Naved Aslam**, Professor, Department of cardiology, **Dr. Bhupinder Singh**, Ass.Professor, Department of cardiology. No part of this thesis has formed the basis for the award of any degree previously.

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Dedicated

to

my Family,

Friends

&

Teachers

ACKNOWLEDGEMENT

As I sit down to pen the conclusion to this thesis, memories flash in my mind and fill my heart with immense gratitude towards all those who with their unending support and encouragement, have contributed in its successful completion and made this an unforgettable experience for me.

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Dr. Akshay Mahajan

LIST OF ABBREVIATIONS

VT	Ventricular tachycardia
SCD	Sudden cardiac death
VF	Ventricular fibrillation
CAD	Coronary artery disease
DCM	Dilated cardiomyopathy
RVOT	Right ventricular outflow tract
LVOT	Left ventricular outflow tract
MI	Myocardial infraction
ECG	Electrocardiography
RFCA	Radiofrequency catheter ablation
ICD	Implantable cardioverter defibrillator
CPR	Cardiopulmonary resuscitation
CAG	Coronary angiography
LVEF	Left ventricular ejection fraction
STEMI	ST elevation myocardial infraction
NSTEMI	Non-ST elevation myocardial infraction
RHD	Rheumatic heart disease
SMVT	Sustained monomorphic ventricular tachycardia
ACLS	Advanced cardiac life support
DVD	Double vessel disease
IABP	Intra-aortic balloon pump
PCI	Percutaneous coronary intervention

CABG	Coronary artery bypass surgery
AICD	Automated implantable cardioverter-defibrillator
CHD	Congential heart disease
NSVT	Non- sustained ventricular tachycardia
ICM	Ischemia cardiomyopathy
SVD	Single vessel disease
TVD	Triple vessel disease
COVID-19	Coronavirus Disease

ABSTRACT

Background: This prospective cohort study was aimed at studying patients presenting with VT to analyze the demographic and hemodynamic features, ECG characteristics, underlying disease, mode of termination and clinical outcome following discharge.

Aims and Objectives: To study the demographical and clinical profile of VT patients. To evaluate the hemodynamics, underlying disease, tachycardia termination and outcome of patients with VT.

Material and Method: This prospective study was conducted at DMCH Ludhiana, a tertiary care center in Ludhiana, India between July 2020 to March 2021. All cases with definite ventricular tachycardia already admitted in the hospital or presenting in emergency department were included in the study. In hospital and 6 month outcome of patient was recorded.

Results: Mean age was 63.5 years and 76/113 of the patients were males. 40.7% were hemodynamically unstable while 59.3% of patients were hemodynamically stable. Coronary artery disease was the most common etiological factor accounting for 54% of patients followed by cardiomyopathy. Determinants of hemodynamic instability were polymorphic pattern of VT, reduced LVEF. In 1.8% cases VT termination was spontaneous, in 51.3% electrical cardioversion was used, drugs were used in 46.9 %.There was reoccurrence of VT episodes in 51%, while 49% patients died at 6 months. Main factor that attributed to recurrence and death was reduced LVEF and presence of CAD.

Conclusion: CAD remains the leading cause of VT. Hemodynamically unstable VT occurs more frequently in polymorphic VT. Most effective method of VT termination is DC cardioversion. CAD, hemodynamic instability and reduced LVEF contributes to overall mortality in VT.

Keywords: VT, CAD, LVEF, Cardioversion

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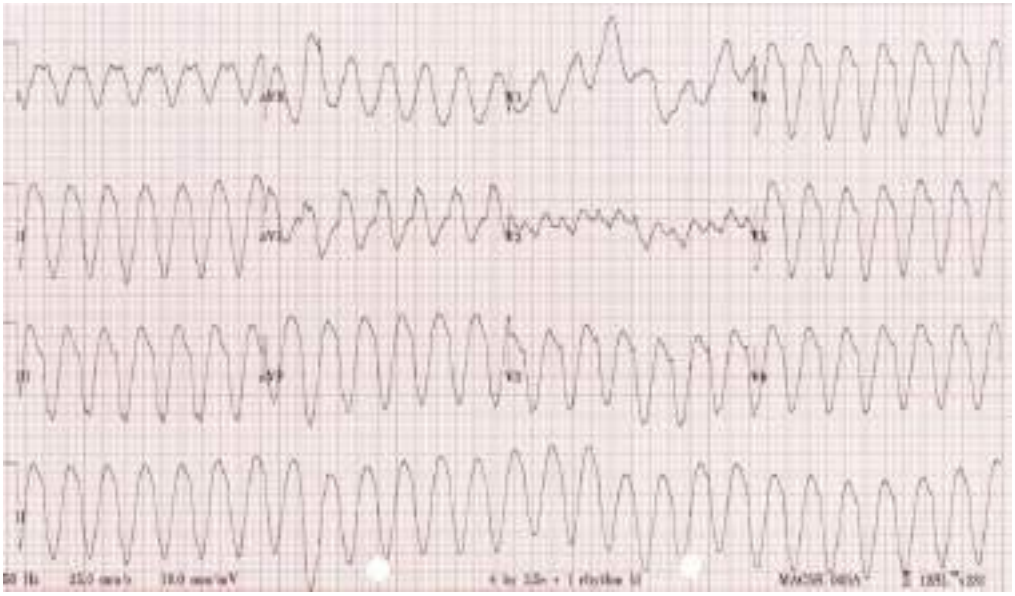
INTRODUCTION

Ventricular tachycardia is a common arrhythmia in patients with structural heart disease and heart failure, and is now seen more frequently as these patients survive longer with modern therapies. Ventricular tachycardia (VT) is defined as a series of three or more ventricular complexes occurring at a rate of 100 to 250 beats per minute. It is the most frequently encountered life threatening arrhythmia.

PICTURE 1 – VENTRICULAR TACHYCARDIA



MAGNITUDE OF THE PROBLEM

Ambulatory ECG recordings at the time of sudden death have shown that, in approximately sixty percent of sudden cardiac death (SCD) victims, an episode of VT was identified as the initial event. [1]

VT is the most common type of broad complex tachycardia, accounting for about eighty percent of all such cases. [2]

EPIDEMIOLOGY

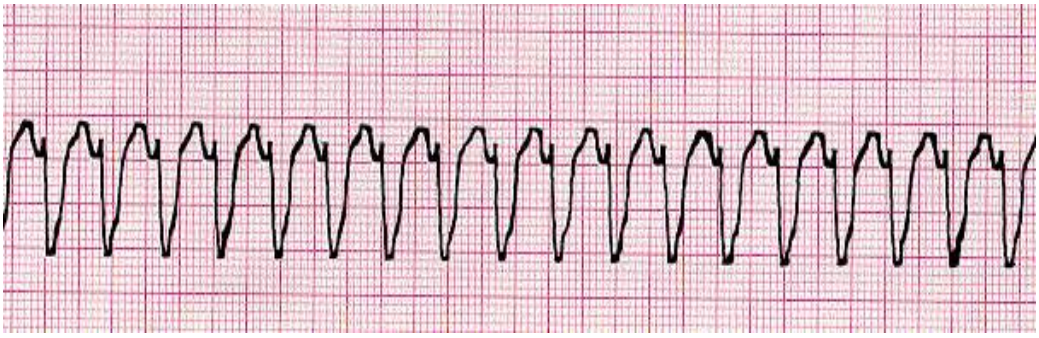
Most sudden cardiac deaths are caused by VT or VF, [3] at an estimated rate of approximately 300,000 deaths per year in the United States, or about half of the estimated cardiac mortality. [4] A prospective surveillance study gave a sudden death incidence of 53 per 100,000

population, accounting for 5.6% of all mortality. [5] This is only a rough estimate of VT incidence, both because many patients have nonfatal VT and because arrhythmic sudden deaths may be associated with VF or bradycardia rather than with VT.

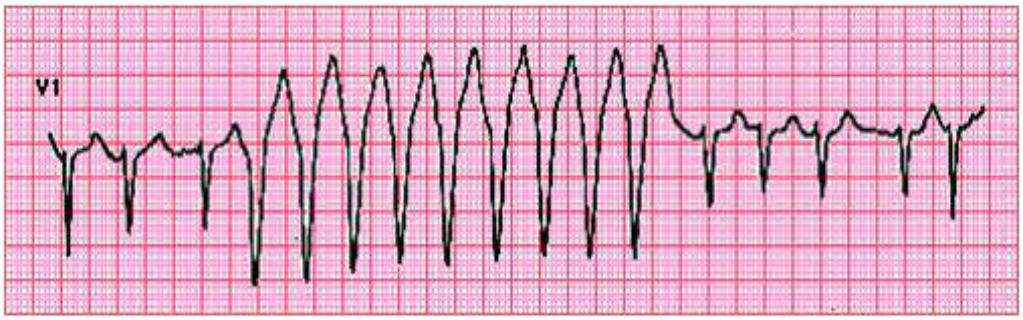
CHARACTERSTIC FEATURES

A time duration of 30 seconds is frequently used to distinguish sustained VT from non-sustained VT. VT requiring immediate intervention is also categorized under Sustained VT.

PICTURE 2- (A) SUSTAINED VENTRICULAR TACHYCARDIA



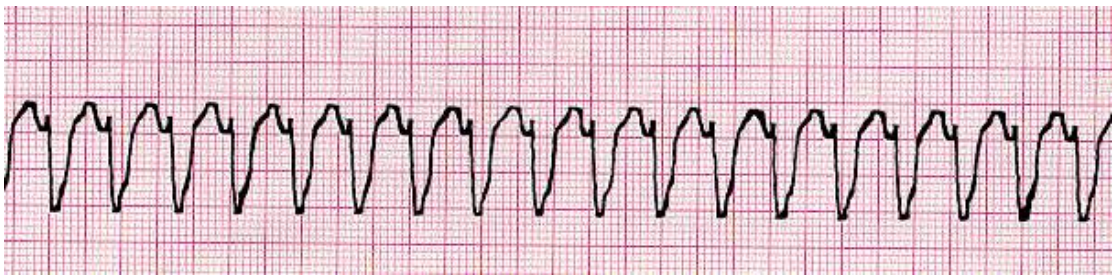
PICTURE 2- (B) NONSUSTAINED VENTRICULAR TACHYCARDIA



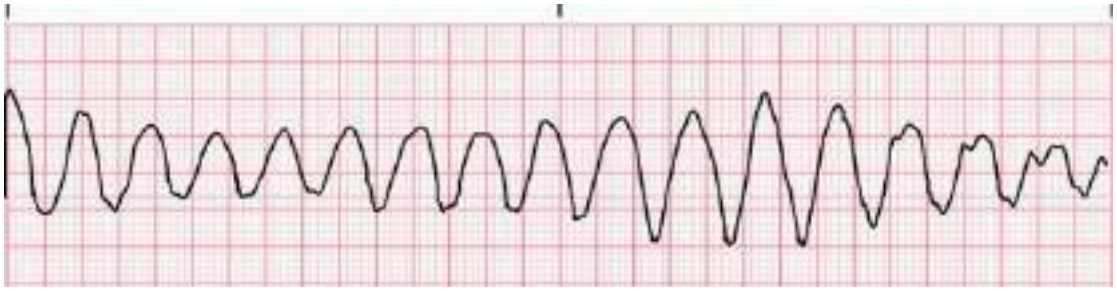
VT can be hemodynamically stable or unstable. Hemodynamic instability is usually observed in patients with underlying heart disease and LV dysfunction. VT degenerating into ventricular fibrillation (VF) is usually fatal.

QRS contours during the VT can be unchanging (monomorphic) or can vary randomly (polymorphic)

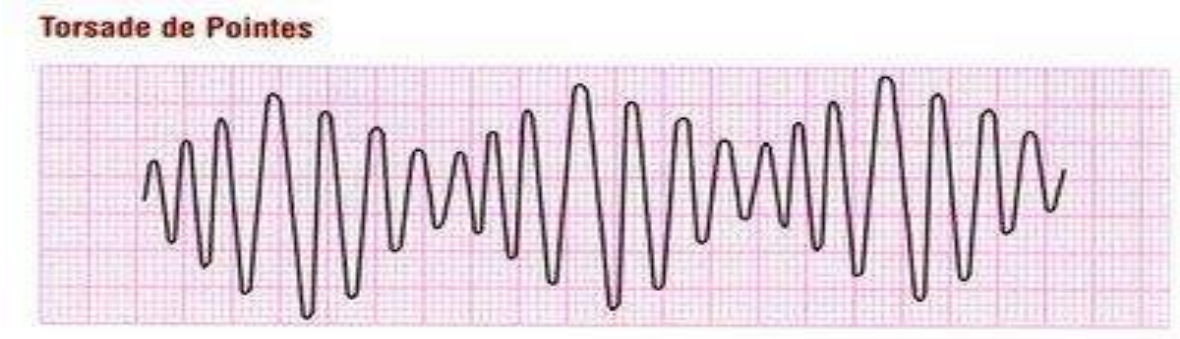
PICTURE 3 - MONOMORPHIC VT



PICTURE 4 - POLYMORPHIC VT



PICTURE 5 - TORSADES DE POINTES



QRS morphology may either show a right bundle branch block (RBBB) or a left bundle branch block (LBBB) pattern. Both morphologies can be seen in the same patient when interventricular septum is involved.

Classification

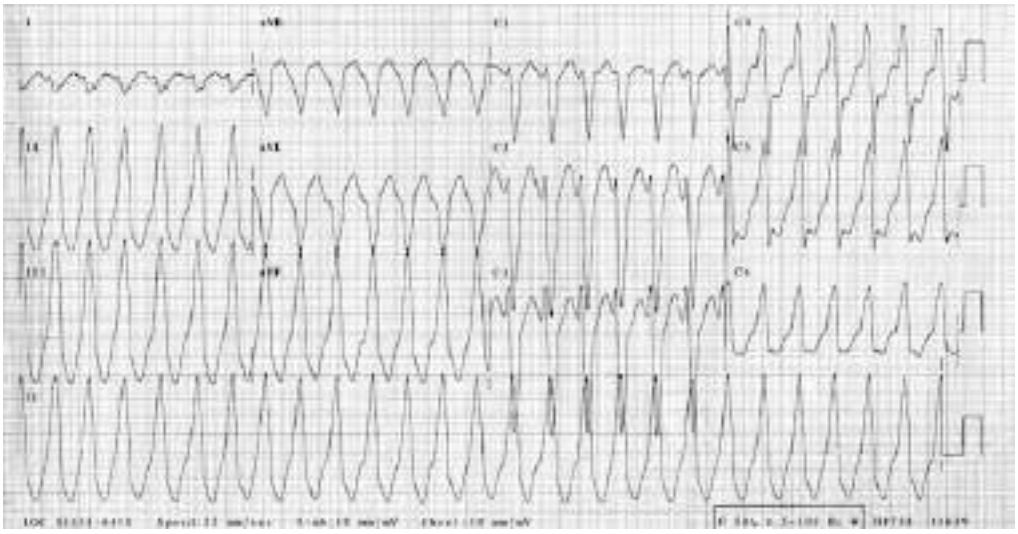
Based upon morphology of the QRS complexes.

Monomorphic Ventricular Tachycardia: In this type QRS complex is uniform. Several distinct types of Ventricular tachycardia are included in this category. There seems to be a relationship between the type of VT and the underlying anatomical substrate. Sustained VT in association with the chronic coronary artery disease (CAD) constitutes the most common type of monomorphic VT seen in the adult

population. [6-8] The typical substrate is within the vicinity of a healed myocardial infarction with or without associated ventricular aneurysm. [9-12] Sustained VT in association with dilated cardiomyopathy (DCM) is another important type in this group .In most of the patients with DCM in association with CAD and in approximately sixty percent of the patients with idiopathic DCM, sustained VT is indistinguishable from that seen in patients with prior myocardial infarction. [13-15] This type of VT can also be seen in patients with no obvious heart disease. [16-18]

Eighty to ninety percent of the Ventricular tachycardia in normal hearts originates from the RVOT and 10% from the LVOT.

PICTURE 6 – RVOT VT



Polymorphic Ventricular Tachycardia: In this type QRS complex varies from beat to beat. Polymorphic VT in patients who demonstrate a long QT interval during their baseline rhythm is referred to as Torsades-de-pointes. Torsades de pointes literally implies a twisting of points (QRS around the baseline) and accurately describes the morphological appearance of this tachycardia. [19-23] It is rapid and polymorphic with a beat-to-beat change in QRS morphology. This type of tachycardia is seen primarily in association with prolongation of the QT interval. The QT (QTc) prolongation could be subtle and dynamic at times.

Both congenital and acquired forms of QT prolongation have been described. [24] Acquired form is more common in adult population and

the QT prolongation in it seems to be mostly in association with the use of agents known to prolong myocardial repolarization, such as class I antiarrhythmic drugs (quinidine, procainamide, disopyramide), amiodarone and in the presence of hypokalemia or hypomagnesemia. The term torsades de pointes is used only when polymorphic VT occurs in the setting of prolonged QT interval whether congenital or acquired. All other non-uniform VT QRS morphologies are classified as polymorphic regardless of how closely they simulate torsades de pointes in terms of QRS appearance. Most, but not all, patients with polymorphic VT in the absence of QT prolongation have CAD. [25] Polymorphic VT is rather an important manifestation of acute myocardial infarction.

PICTURE 7 – Bidirectional Ventricular Tachycardia

Bidirectional Ventricular Tachycardia: VT with beat to beat changing QRS frontal plane axis, indicator of digoxin toxicity or catecholaminergic polymorphic VT.

Aetiology

1. VT is often a symptom of CAD or other structural heart diseases. More than 50% of patients treated for VT have

ischaemic heart disease. The overall incidence of sustained VT following MI was classically established at about three to five percent but has declined to one percent in recent years due to major advances in MI management, resulting in smaller infarct scars. The VT risk in overall population however has been fairly stable and could infact be increasing, on account of increased post MI survival and the possibility of VT years after the initial MI. [26] Although this type of re- entrant monomorphic VT has been primarily studied in patients of CAD, other cardiac pathologies resulting in critical amount of myocardial fibrosis may be associated with similar arrhythmias. [27]

2. Valvular heart disease and myocarditis can also lead to VT.
3. Cardiomyopathies constitute the next largest group which includes
    - Hypertrophic cardiomyopathy
    - Dilated cardiomyopathy
    - Infiltrative cardiomyopathy
    - Arrhythmogenic right ventricular cardiomyopathy
4. VT can be triggered by serum electrolyte deficiencies like hypocalcemia, hypomagnesemia and hypokalemia.
5. Catacholaminergic agents, class 3 anti-arrhythmic drugs and digoxin toxicity can also lead to VT.
6. After surgical revascularization and after surgical treatment of Tetrology of Fallot.
7. Neuromuscular diseases like
    - Sarcoidosis
    - Chagas disease
    - Amyloidosis
    - Fabry’s disease
    - Hemochromatosis
    - Friedrich’s ataxia

8. Brugada syndrome: It can be inherited and generally affects males. It shows manifest or concealed ST segment elevation in V_1-V_3 and right bundle branch block.
9. Long QT syndrome.
10. Idiopathic.

PATHOGENESIS

The pathogenesis of VT depends upon its aetiology and type. In monomorphic VT the impulse is either being generated from increased automaticity of a single point in either left or right ventricle, or due to a re-entry circuit in the ventricle. From clinical studies as well as animal models of VT in association with myocardial infarction (MI), it can be reasonably assumed that the underlying mechanism is re- entry. Re-entry is the mechanism underlying the VT associated with healed or healing MI in more than ninety five percent of cases. The most common cause of monomorphic VT is myocardial scarring from a previous MI. The scar cannot conduct electricity so there is a potential circuit around the scar that results in tachycardia. In case of idiopathic dilated cardiomyopathy, more than one third of the patients have sustained Bundle Branch Re-entry as the cause of monomorphic VT.[28]

The precise mechanism for torsade-de-pointes is not known. However, recent data strongly suggests that triggered activity due to early after depolarizations may be playing an important role in the acquired form.

The predisposition to this problem usually manifests on the ECG as a prolongation of QT interval which can be congenital or acquired.

PRESENTATION

The clinical presentation of the VT depends on the hemodynamic consequences it produces.

1. Physical examination of a patient presenting with VT often indicates hemodynamic distress (low blood pressure, heart failure, or cardiogenic shock). Patient can have
    - Fainting (syncope)
    - Loss of consciousness

- Pulselessness

2. When cardiac output and blood pressure are maintained, VT presents as
    - Chest pain (angina)
    - Palpitations
    - Breathlessness
3. Among all cardiac arrest patients, syncope and presyncope are the most common manifestations. Syncope is the single most important clinical event for grading sudden cardiac death risk in heart failure patients. VT was found to be cause of syncope in 35% of these patients. [29]
4. In patients with significant CAD or left ventricular dysfunction, rapid degeneration to ventricular fibrillation (VF) may occur.

TREATMENT

The management of VT is multimodal involving the use of antiarrhythmic drugs (class 1 & 3), DC cardioversion, radiofrequency catheter ablation (RFCA), correction of electrolyte abnormalities and the treatment of underlying aetiology. High risk patients are treated with implantable cardioverter-defibrillator (ICD). [30-35]

AIMS AND OBJECTIVES

- To study the demographical profile of Ventricular Tachycardia patients.
- To study the clinical profile of Ventricular Tachycardia patients.
- To evaluate the hemodynamic, underlying disease, tachycardia termination and outcome of patients with ventricular tachycardia.

REVIEW OF LITERATURE

VT is a life threatening arrhythmia which needs prompt recognition and management to save the life.

With this background, we studied the clinical spectrum and outcome of patients of VT reporting in our institution.

Akhtar M (1990) [36] studied the spectrum of VT and observed that CAD is the most common cause of sustained monomorphic VT. Polymorphic VT is caused by both acquired and congenital forms of QT prolongation. The acquired form of QT prolongation is seen in association with the use of antiarrhythmic drugs and in the presence of hypokalemia and hypomagnesemia. Most but not all the patients with polymorphic VT in the absence of QT prolongation have CAD.

Domanovits H, Paulis M, NikfardJam M et al (1999) [37] studied sustained VT in 75 patients in the emergency department and observed that ischaemic heart disease was underlying disorder in majority of the patients (76%). Hemodynamic instability was present in 23% patients. Polymorphic VT was present in 9% of patients. Non sustained VT was present in 4% patients. Mortality was 7%. Majority of the patients (88%) were initially treated with anti-arrhythmic medication, but half of these patients needed DC cardioversion finally for the definitive termination of VT. Thus 50% of patients needed both medical management and DC shock for management of VT.

Elliot PM, Sharma S, Varnava A (1999) [38] studied survival after sustained VT in patients with hypertrophic cardiomyopathy and found that the patients with hypertrophic cardiomyopathy who survive an episode of VT remain at risk for recurrent event. Implantable cardioverter-defibrillator (ICD) therapy appears to offer the best potential benefit regarding outcome.

Steinberg JS, Gaur A et al (1999) [39] studied new onset sustained VT after cardiac surgery and found that the first presentation of sustained monomorphic VT in the recovery period after coronary artery bypass

grafting (CABG) is uncommon, but the incidence is high in specific clinical subsets. Placement of a bypass graft across a non-collateralized total occlusion vessel supplying an infarct zone was strongly and independently associated with the development of VT.

Kautzner J (2000) [40] studied clinical spectrum of VT and observed that among the monomorphic VT, the sustained VT after myocardial infarction is the most frequent form, accounting for more than 70% of all VT cases.

Pires LA, Lehmann MH, Buxton AE et al (2001) [41] studied inducibility and prognosis of in-hospital versus out of hospital identified non sustained VT (NSVT) in patients with CAD and found that in patients with CAD and impaired left ventricular ejection fraction (LVEF), asymptomatic NSVT identified in hospital, compared with that identified out of hospital is associated with higher rate of induction of sustained VT and overall mortality, therefore in similar patients, the clinical setting in which non sustained VT is discovered should be taken in to consideration while formulating patient risk and treatment.

Nakagawa M, Takashashi N, Nobe S et al (2002) [42] studied gender differences in various types of VT and observed that gender specific differences exist in incidence and age distribution of various types of idiopathic VT.

Di Lenarda A, Pinamonti B, Mestroni L et al (2004) [43] studied the natural history of dilated cardiomyopathy (DCM) in 581 patients and observed that long term optimised treatment with ACE inhibitors (in 90% of cases) and beta blockers (in 87% of cases) was associated with a remarkable clinical improvement in 50% of the patients and apparent healing in 16% of the cases. The risk of sudden cardiac death (SCD) was increased particularly in the patients with long term persistant or progressive left ventricular dilation and dysfunction.

Cuoco FA, Singh SN (2005) [44] studied NSVT in dilated cardiomyopathy (DCM) and observed that NSVT is common in patients with DCM of both ischaemic and non ischaemic origin. Recent studies

suggest that NSVT may be a marker but not a significant predictor of mortality and sudden cardiac death (SCD).

Harris KC, Potts JE et al (2006) [45] studied right ventricular outflow tract tachycardia (RVOT) in children and observed that clinical spectrum and management of RVOT in children are diverse. Both medical therapy and observation alone were associated with a reduction in ectopy.

Varriale P, Leonardi M (2006) [46] studied polymorphic VT in the coronary care unit and observed that acute myocardial infarction is the most common cause of polymorphic VT associated with a normal QT interval . Polymorphic VT with or without QT prolongation is a sporadic tachyarrhythmia that has frequent malignant potential for cardiac arrest. Successful management mandates emergency defibrillation for cardiac arrest and other appropriate measures for suppression of polymorphic VT. The prognosis of polymorphic VT is improved when the cause is correctly identified and promptly treated.

Kanjwal K, Imraan N, Grubb B (2008) [47] studied the troponin elevation in patients with various tachycardias and normal epicardial coronaries and observed that the patients with elevated troponin due to conditions other than acute coronary syndrome can receive inappropriate and delayed definitive diagnosis and treatment. So, in the patients admitted within the frame work of tachyarrhythmia without hemodynamic instability, the decision whether to obtain serum markers of myocardial ischaemia should cover a comprehensive clinical context.

Suesaowalak M, Khongphattanayothiu A et al (2008) [48] studied idiopathic left ventricular tachycardia in children and found that median age at presentation was 9.5 years. Majority of the patients responded to intravenous verapamil.

Aleksova A, Sabbadine G et al (2009) [49] studied the frequency with which the patients effected by the dilated cardiomyopathy (DCM) are diagnosed in the asymptomatic state and factors influencing their

outcome. They observed that the proportion of asymptomatic patients with DCM at the moment of first evaluation is 30%. Among these, the patients without a previous history of heart failure had a less advanced disease and a trend for better long term quality of life and even better survival.

Pascale P, Schlapter J, Oddo M et al (2009) [50] studied VT in CAD and observed that inferior scars may be more prone to develop VT as compared to anterior scars.

Kjeldsen K (2010) [51] studied hypokalemia and sudden cardiac death (SCD) and observed that hypokalemia is present in 7 % to 17% of the patients with cardiovascular disease. Up to 40% of the patients on diuretics suffer from hypokalemia. Inadequate management of hypokalemia was found in 24% of hospitalized patients. Hypokalemia is associated with increased risk of cardiac arrhythmia in patients with cardiovascular disease as well as increased all-cause mortality by up to ten fold.

Mazic S, Lazovic B, Delic M et al (2012) [52] worked on the arrhythmogenic right ventricular dysplasia (ARVD) as a cause of sudden cardiac death (SCD) in the young people and found that the incidence of ARVD is about 1/1000 to 1/5000 and the presentation is usually related to VT, syncope, presyncope or ventricular fibrillation (VF) leading to cardiac arrest. It occurs mostly in young people and athletes. The intracardiac defibrillator (ICD) is the most effective safeguard against sudden cardiac death (SCD).

Mehta RH, Yu J, Piccini JP et al (2012) [53] studied the prognostic significance of post procedure sustained VT in patients undergoing primary percutaneous coronary intervention (PPCI) for ST elevation MI (STEMI) and found that out of 3485 patients undergoing PPCI in whom VT/VF did not develop before or during procedure, 5.2% developed VT/VF after PPCI. Most VT/VF occurred in the first forty eight hours after procedure. Majority of these patients were killip class >1 on presentation.

Nerry PB, Leung E et al (2012) [54] studied arrhythmias in patients of sarcoidosis and proposed that sarcoidosis should be considered in patients aged less than fifty five years presenting with unexplained AV block and in patients with sustained VT.

Agir A, Bozyel S, Celikyurt u et al (2014) [55] studied ARVD in pregnancy and observed that pregnancy is well tolerated in women affected by ARVD and can be managed successfully with close monitoring and anti-arrhythmic drugs when necessary. ICD implantation should also be considered to prevent sudden cardiac death (SCD) during pregnancy to prevent life of mother. Pregnancy seems not to cause an increase in major ICD related complications.

Sousa PA, Pereira S et al (2014) [56] studied the value of electrocardiography for differential diagnosis of wide QRS complex tachycardias and observed that in small percentage of wide QRS complex tachycardias, the diagnosis remains un-certain and in these cases the wisest decision is to treat them as VT.

DeSouza IS, Martindale JL, Sinert R (2015) [57] performed a systematic review of the literature to compare the efficacy of different drug therapies for the termination of stable, monomorphic ventricular tachycardia. Based on limited available evidence from small heterogeneous human studies, for the treatment of stable, monomorphic VT, procainamide, ajmaline and sotalol were all superior to lidocaine; amiodarone was not more effective than procainamide.

Behere SP, Weindling N (2015) [58] review of the current literature on CPVT aims to summarize the state of the art in our understanding of the genetic etiology and the molecular pathogenesis of CPVT, and how these relate to our current approach to diagnosis and management. We will also shed light on groundbreaking new work that will continue to refine the management of CPVT in the future. As our knowledge of CPVT continues to grow, further studies will yield a better understanding of the efficacy and pitfalls of established diagnostic approaches and therapies as well as help shape newer diagnostic and

treatment strategies. Two separate searches were run on the National Center for Biotechnology Information's (NCBI) website.

Chin-Yu Lin, Shih-Lin Chang et al (2016) [59] studied impact of non-sustained ventricular tachycardia (NSVT) on the risk of thromboembolic event and clinical outcomes in patients without structural heart disease remains undetermined. This study aimed to evaluate the association between NSVT and clinical outcomes and it was found that in patients without structural heart disease, presence of NSVT on 24-hour monitoring was independently associated with death, CV hospitalization, ischemic stroke, TIA and new onset heart failure.

Kumar S, Romero J et al (2016) [60] did study to describe long-term prognosis after VT ablation in patients with no structural heart disease (no SHD), ischemia cardiomyopathy (ICM) and non-ischemic cardiomyopathy (NICM) and it was found that Long-term follow-up after VT ablation shows excellent prognosis in the absence of SHD, highest VA recurrence, and transplantation in patients with NICM and highest mortality in patients with ICM. The extremely low mortality for those without SHD suggests that VT in this population is rarely an initial presentation of a myopathic process.

Mahida S, Sacher F et al (2017) [61] provides an overview of the role of cardiac imaging in patients with VT. Ventricular tachycardia (VT) is a major cause of sudden cardiac death. The majority of malignant VTs occur in patients with structural heart disease. Multimodality imaging techniques play an integral role in determining the underlying etiology and prognostic significance of VT. In recent years, advances in imaging technology have enabled characterization of the structural arrhythmogenic substrate in patients with VT with increasing precision. In parallel with these advances, the role of cardiac imaging has expanded from a largely diagnostic tool to an adjunctive tool to guide interventional approaches for treatment of VT. Invasive and non-invasive imaging techniques, often used in combination, have made it possible to integrate structural and electrophysiological information

during VT ablation procedures. An important area of current development is the use of non-invasive imaging techniques based on body surface electrocardiographic mapping to elucidate the mechanisms of VT. In the future, these techniques may provide a priori information on mechanisms of VT in patients undergoing interventional procedures.

Sudan R, Yaqoob L et al (2018) [62] studied the Profile of patients presenting with sustained ventricular tachycardia in a tertiary care center. It was concluded that Ischemic heart disease remains the leading cause of VT. Hemodynamically unstable VT occurs more frequently in acute myocardial infarction and polymorphic VT. Most effective method of VT termination is DC cardioversion. Old age, hemodynamic instability and ejection fraction contribute to overall mortality in VT.

Viskin S, Chorin E et al (2019) [63] Polymorphic ventricular tachycardia (VT) without QT prolongation is well described in patients without structural heart disease (mainly idiopathic ventricular fibrillation and Brugada syndrome) and in patients with acute ST-elevation myocardial infarction and it was concluded that Arrhythmic storm with recurrent polymorphic VT in patients with coronary disease responds to quinidine therapy when other antiarrhythmic drugs (including intravenous amiodarone) fail.

Yang J, Brunnquell M et al (2019) [64] studied that ventricular tachycardia (VT) is frequently encountered in patients with repaired and unrepaired congenital heart disease (CHD), causing significant morbidity and sudden cardiac death. Data regarding underlying VT mechanisms and optimal ablation strategies in these patients remain limited .it was concluded that while scar-related re-entry is the most common VT mechanism in patients with CHD, importantly, non scar-related VT may also be present. In experienced tertiary care centers, ablation of both scar-related and non scar-related VT in patients with CHD is safe, feasible, and effective over long-term follow-up.

Havakuk O, Viskin D et al (2020) [65] did clinical presentation of sustained monomorphic ventricular tachycardiawithout cardiac Arrestand itwas concluded that despite similar heart rate, patients with VT rarely report having palpitations, whereas patients with SVT do socommonly.This finding may assist with decision making in patients reporting palpitations in whom an ECG tracing is not available.

Kallas D, Roston TM et al (2021) [66] studied to determine whether proband status, age at symptom onset, and/or sex are independent predictors of cardiac events in pediatric catecholaminergic polymorphic vt and it was concluded that proband status, but not age at symptom onset or male sex, independently predicted an earlier onset of cardiac events. A larger sample size would enable a comprehensive investigation of other risk factors.

MATERIALS AND METHODS

All the patients admitted in the cardiology department of DMC hospital with diagnosis of VT on ECG.

METHOD OF COLLECTION OF DATA

Inclusion Criteria:

All cases with definite ventricular tachycardia already admitted in the hospital or presenting in emergency department were included in the study.

Exclusion Criteria:

1. Patients without known etiology who died early after hospitalization thereby precluding further evaluation.
2. Patients who refused for further evaluation.
3. Patients with recurrent episodes of VT, only the first episode was considered as index event and evaluated.
4. Patients with out of hospital pulse less VT that was terminated before arrival to our hospital but had no documentation of the same.
5. Patients with pre-implanted implantable cardioverter defibrillator (ICD) because of the different management of such group.

Study design

Prospective observational study

Study place

Department of cardiology, DMCH, Ludhiana

The study was conducted in the Department of Cardiology of DMCH, Ludhiana. It was a time based study conducted between July 2020 to March 2021. Total-129 patients were initially included however 16 patients were lost to follow up, so total 113 patients were taken into the study.

A detailed history pointing to the possible aetiology of VT was obtained from all patients. It included history of CAD, heart failure, hypertension, cardiomyopathies, other associated risk factors drug intake, family history and drugs given at discharge.

Patients were subjected to physical examination. Pulse, blood pressure and features of heart failure were recorded to assess hemodynamic status.

These patients were divided into two sub-groups: hemodynamically stable and hemodynamically unstable. Hemodynamic unstability is defined as need for immediate intervention due to loss of consciousness, hypotension or occurrence of congestive heart failure. Otherwise patients were defined as hemodynamically stable.

Relevant investigations carried out included:

12 LEAD ECG: ECG of all the patients was seen satisfying the standard criterion of VT by applying the Brugada algorithm and/or the aVR Vereckei algorithm. In cases of non-sustained VT, ECG recordings were seen from the bedside monitors attached to the patient or 24 hour holter recordings.

ECHOCARDIOGRAPHY: Echocardiography was done in all patients of VT to see their left ventricular ejection fraction (LVEF) and various other dimensions were also noted. Patients having LVEF of less than 40% were classified as having low LVEF. Further Echocardiography was used to see any underlying cardiac disease to help in arriving at possible aetiology.

SERUM ELECTROCYTES:

A. **POTASSIUM**: Potassium levels were seen in all the patients to look for hypokalemia. Hypokalemia as defined by potassium levels of less than 2.7 m Mol / litre was taken in to consideration. Patients having hypokalemia without any other underlying cardiac disease as well as those having hypokalemia contributing to some other aetiology of VT were considered.

B. **MAGNESIUM**: Magnesium levels were seen in all the patients to look for hypomagnesaemia. Hypomagnesaemia as defined by magnesium levels of less than 1.7 m Mol/litre was taken in to consideration.

Complete hemogram, Serum urea, creatinine and liver function tests were seen in all the patients as a part of routine investigations.

SPECIAL INVESTIGATIONS: Done in specific cases included

CORONARY ANGIOGRAPHY (CAG):- Patients who were known cases of coronary artery disease (CAD) presenting with features of VT as well as those who presented as CAD and developed VT as a complication in the same admission were included in the study. Thus VT occurring as an early as well as late complication of CAD was seen.

VT termination and follow-up:-

The therapy for termination of VT protocol was as per latest ACLS guidelines. Patients who were hemodynamically unstable were administered electrical cardioversion. 100J of synchronized direct current (DC) was given under proper sedation and analgesia. The energy was subsequently increased to a maximum of 200J wherever required. Pulse less VT patients were administered cardiopulmonary resuscitation CPR as per ACLS guidelines. Intravenous antiarrhythmic drugs in recommended doses were given to hemodynamically stable patients. The choice of antiarrhythmic drug was based on attending physician’s discretion. Electrical cardioversion was given to those patients who became secondarily unstable or in whom antiarrhythmic drugs failed to terminate VT.

Interventions done in specific aetiologies such as PCI, CABG and AICD were also recorded.

6 months Follow-up information of each patient regarding recurrence or death was obtained.

Statistical analysis

Statistical analysis was performed by SPSS software package (version 20.0, SPSS Inc, Chicago, Illinois, USA). All continuous variables were expressed as Mean, SD and categorical variable was reported as frequency and percentages. Group comparisons was performed with Student t-test or crosstabs. The Pearson chi-square test or Fisher exact test was used for categorical variables. A p-value of <0.05 was considered statistically significant.

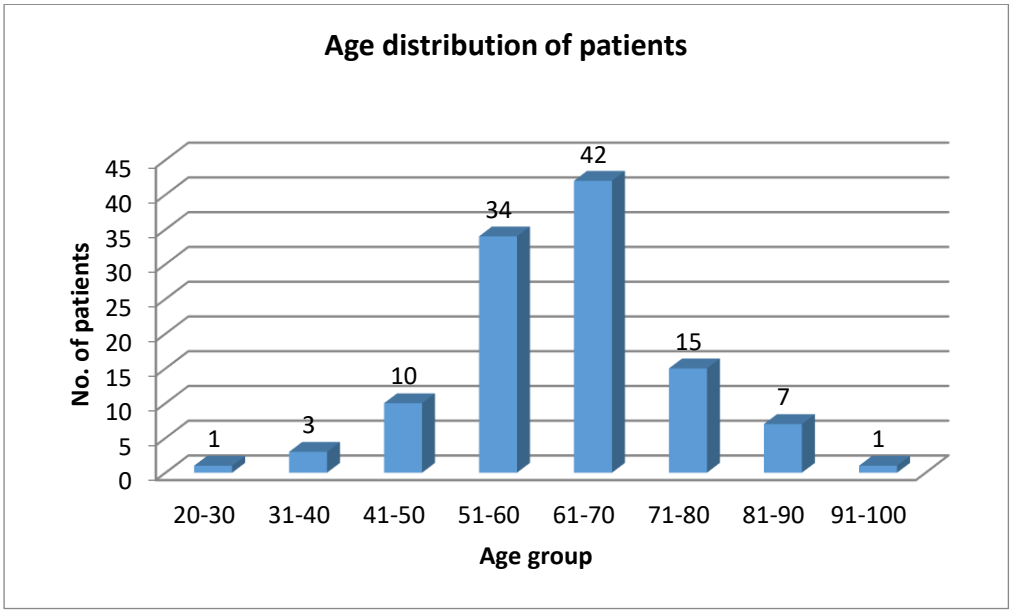
OBSERVATION AND RESULTS

Table 1: AGE AND SEX DISTRIBUTION OF DIFFERENT PATIENTS STUDIED

Category	Mean	N	Std. Deviation
Female	62.73	37	9.380
Male	63.21	76	12.578
Total	63.05	113	11.587

As is evident from above table, the mean age of patients developing VT was 63.21 years in case of males and it was 62.73 years in case of females.

FIGURE 1: AGE DISTRIBUTION OF PATIENTS



In our study, out of 113 patients maximum patients were in age group 51-70 years and minimum patients were in age group <30 or >90 years.

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Table 2: DISTIBUTION OF PATIENTS ACCORDING TO CAUSES OF VT

CAUSES OF VT	No. of patients	Percent
CAD	61	54.0
Cardiomyopathy	32	28.3
RHD	7	6.2
Congenital	7	6.2
Diselectrolytemia	6	5.3
Total	113	100.0

In maximum cases CAD was the major cause of VT (54%) followed by cardiomyopathy seen in 28.3%, rheumatic heart disease and congenital cause was seen in 6.2%, 5.3% showed diselectrolytemia.

Figure 2: DISTRIBUTION OF PATIENTS ACCORDING TO CAUSES OF VT

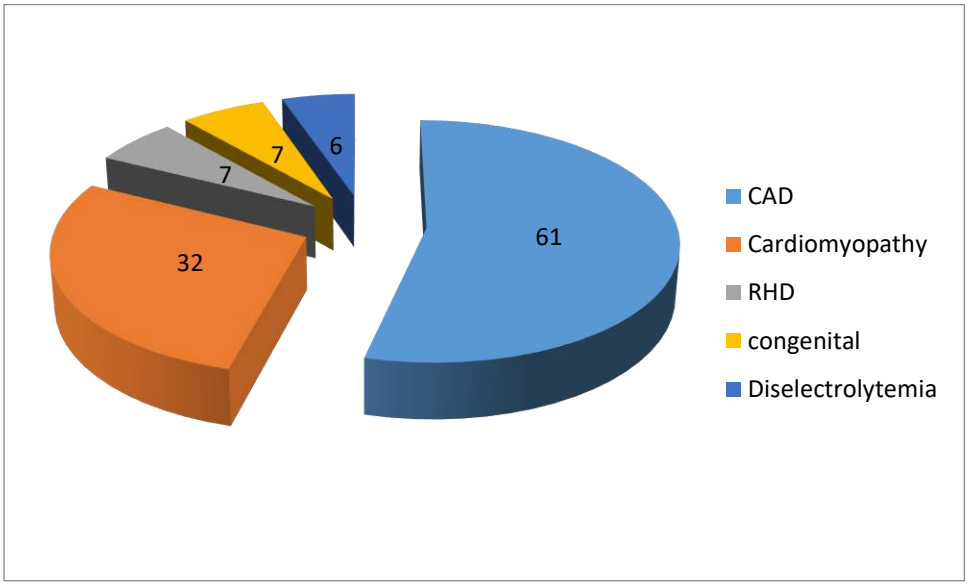


TABLE 3: CHIEF COMPLAINTS PRESENTED BY PATIENTS AT THE TIME OF ADMISSION

Chief complain	No. of patients	Percentage of patients
Chest pain	66	58.4
Shortness of breath	47	41.6
Palpitations	12	10.7
Syncope	9	8
Dizziness	15	13.3
Fatigue	2	1.8
Presyncope	0	0

In present study maximum patients presented with chest pain (58.4%) followed by shortness of breath (41.6%) dizziness (13.3%) palpitations (10.7%), syncope (8%) and fatigue (1.8%). However at the time of VT main chief complaints were dizziness and palpitations.

FIGURE 3: DISTRIBUTION OF PATIENTS ACCORDING TO CHIEF COMPLAINTS AT TIME OF ADMISSION

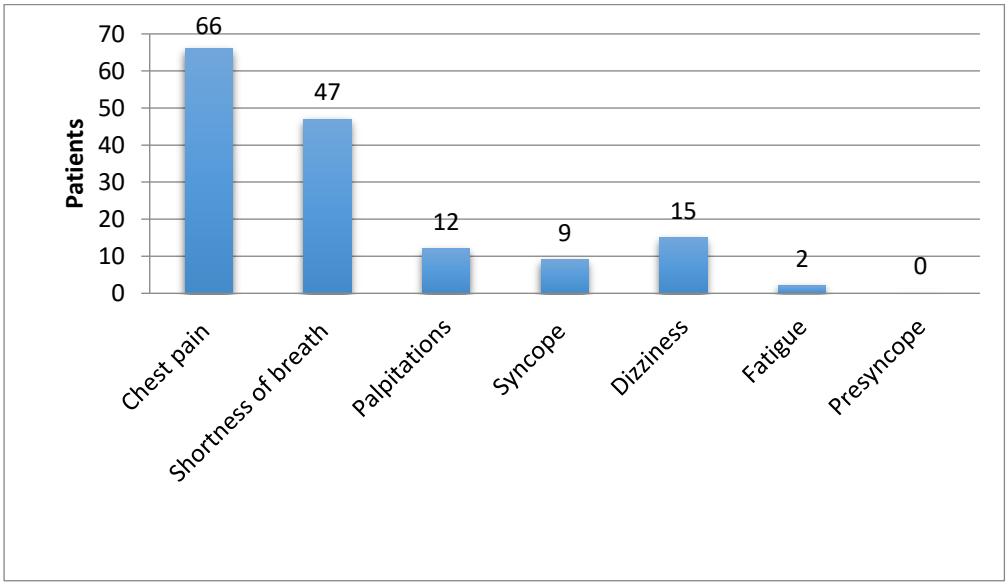


TABLE 4: DISTRIBUTION OF PATIENTS ACCORDING TO ASSOCIATED RISK FACTORS

Risk Factors	No. Of Patients	Percentage Of Patients
Diabetes	38	33.6
Hypertension	27	23.8
Smoking	21	18.6
Thyroid Disorder	5	4.4
Bronchial Asthma or tuberculosis	2	1.8
CVA	1	0.9

It was observed that maximum patients had risk factors of Diabetes (33.6%), followed by hypertension (23.8%) , Smoking (18.6%), thyroid disorder (4.4%), Bronchial Asthma or tuberculosis (1.8%) and CVA (0.9%).

FIGURE 4: DISTRIBUTION OF PATIENTS ACCORDING TO ASSOCIATED RISK FACTORS

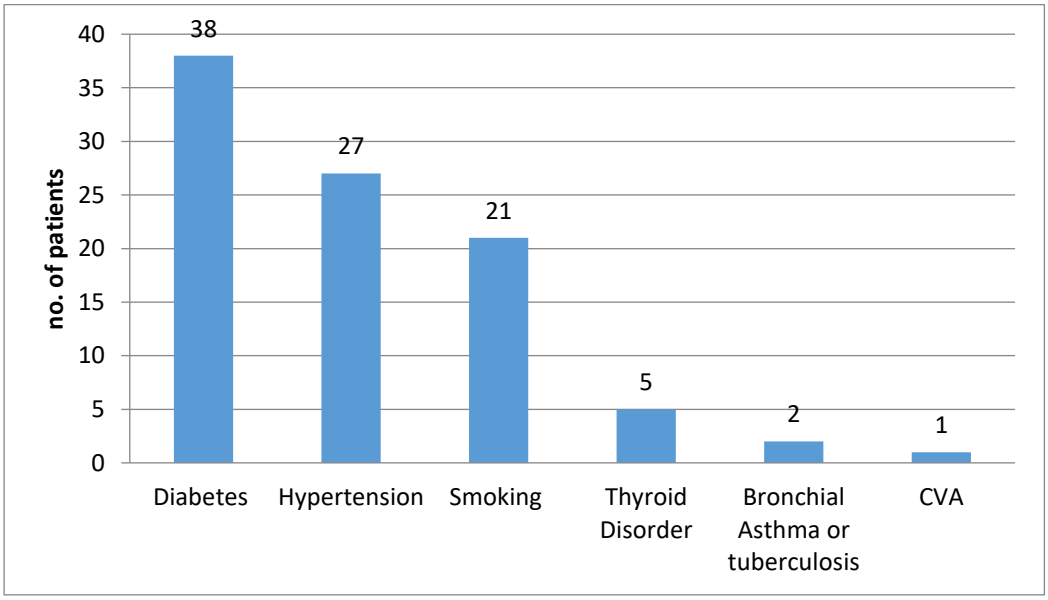


TABLE 5: DISTRIBUTION OF PATIENTS ACCORDING TO CAD

CAD finding	Frequency	Percent
NSTEMI	25	22.1
STEMI	26	23
Angina	10	8.8
Total	61/113	100

Among 61 patients of CAD 25/61 were of NSTEMI 26/61 were of STEMI and 10/61 cases were of angina

FIGURE 5: DISTRIBUTION OF PATIENTS ACCORDING TO CAD

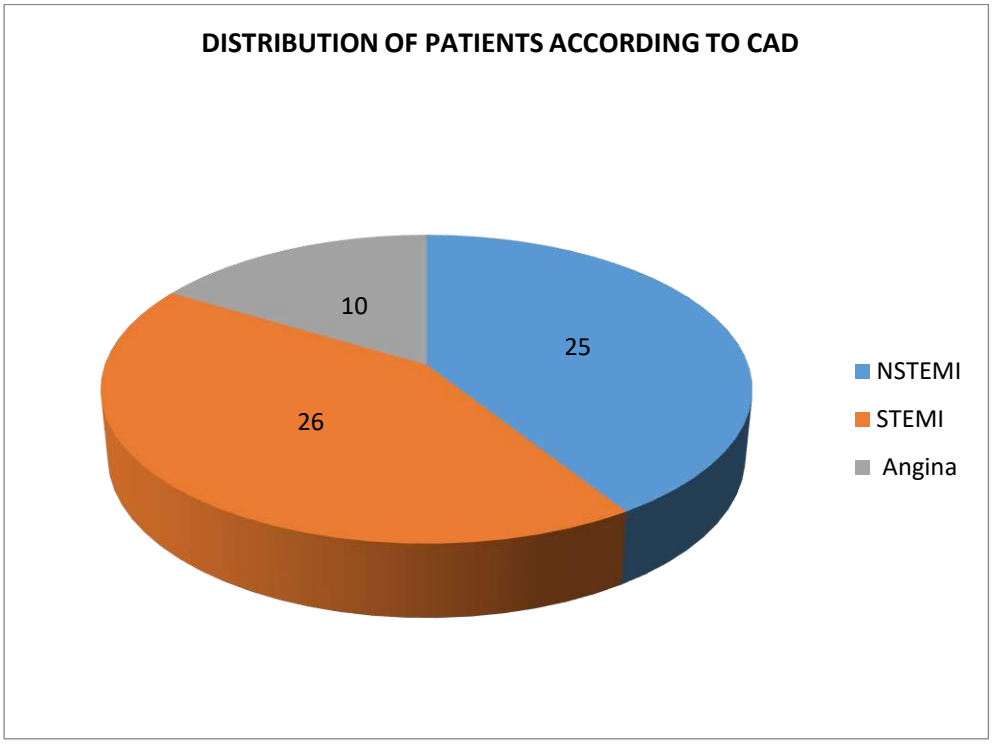


TABLE 6: DISTRIBUTION OF PATIENTS ACCORDING TO HEMODYNAMIC STABILITY

HEMODYNAMICS	No. of patients	Percentage
Stable	67	59.3
Unstable	46	40.7
Total	113	100.0

Out of 113 patients studied, 46 patients (40.7%) were unstable hemodynamically. 67 (59.3%) of patients were hemodynamically stable.

FIGURE 6: DISTRIBUTION OF PATIENTS ACCORDING TO HEMODYNAMIC STABILITY

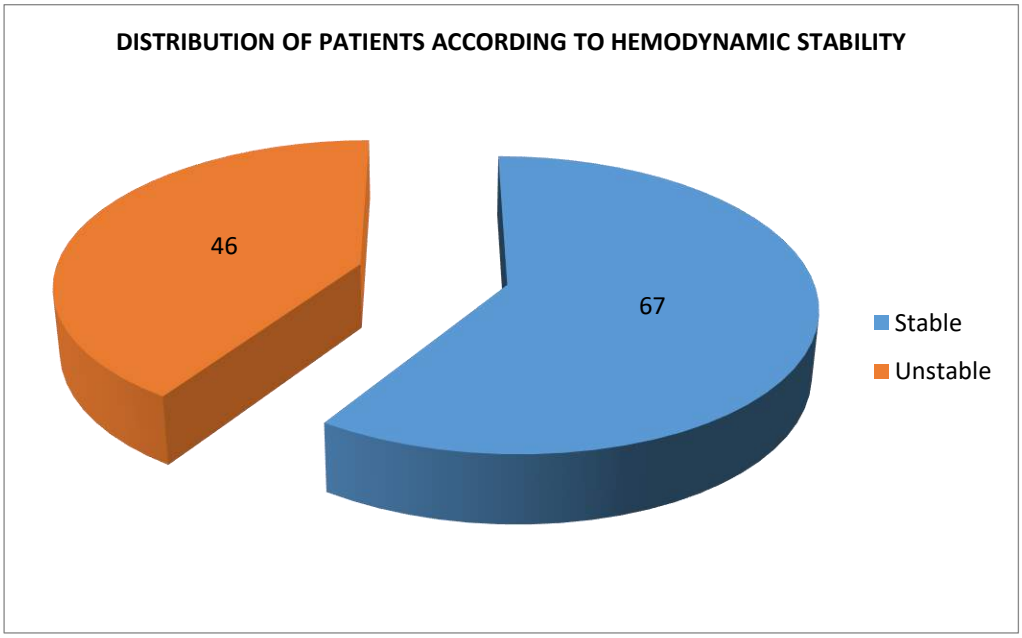


TABLE 7: DISTRIBUTION OF PATIENTS ACCORDING TO VT MORPHOLOGY

MORPHOLOGY	No. of patients	Percentage
MONOMORPHIC	71	62.8
POLYMORPHIC	42	37.2
Total	113	100.0

Out of 113 patients studied, 71 patients (62.8%) were having monomorphic VT while 42% (37.2%) have polymorphic VT.

FIGURE 7: DISTIBUTION OF PATIENTS ACCORDING TO VT MORPHOLOGY

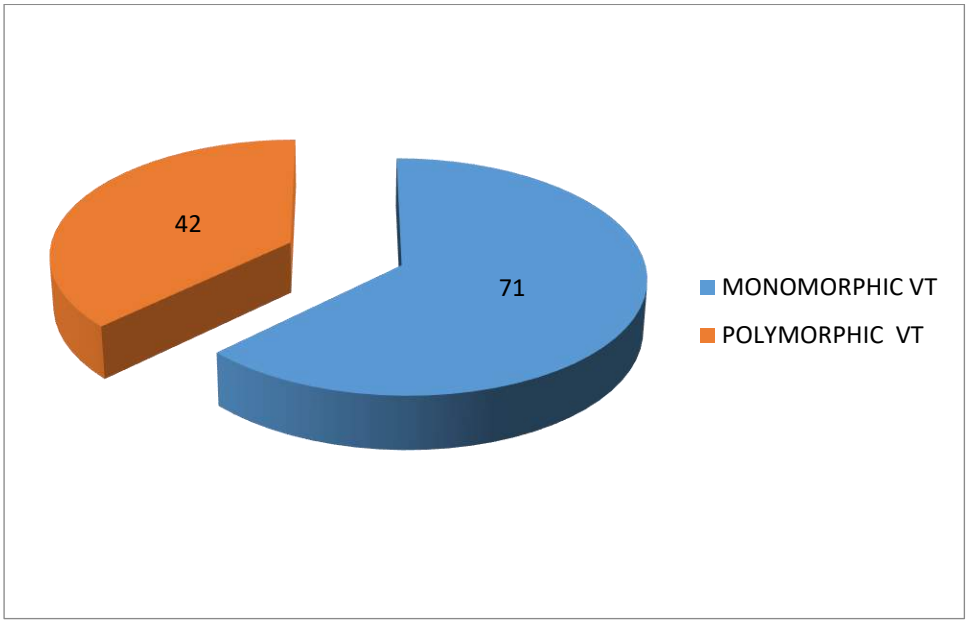


TABLE 8: DISTRIBUTION OF CASES ACCORDING TO OCCURRENCE OF VT

VT occurrence	Frequency	Percent
At the time of admission	11	9.7
Within 24 hours	65	57.6
After 24 hours	37	32.7
Total	113	100.0

In 9.7% VT occurred at the time of admission in 57.6% cases VT occurred within 24 hours of admission while in 32.7% cases it occurred after 24 hours.

FIGURE 8: DISTRIBUTION OF CASES ACCORDING TO OCCURRENCE OF VT

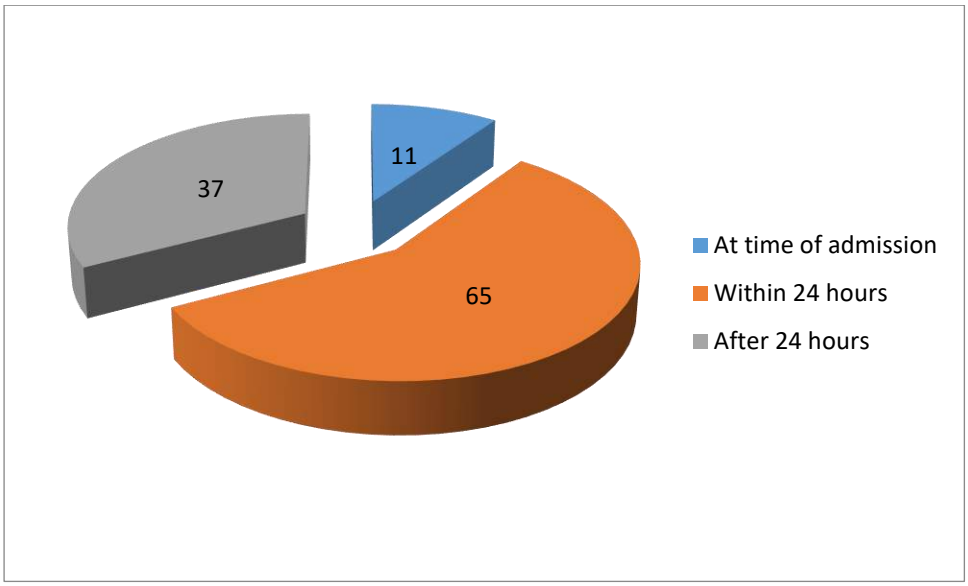


TABLE 9: DISTRIBUTION OF CASES ACCORDING TO VT TERMINATION

VT termination	Frequency	Percent
Spontaneous	2	1.8
Drugs (amiodarone, lignocaine, others)	53	46.9
Electrical Cardioversion	58	51.3
Total	113	100.0

In 1.8% cases VT termination was spontaneous, in 51.3% electrical cardioversion was used, drugs were used in 46.9 %.

Out of all 58 patients who were given electrical cardioversion, 15/58 (13.27%) were cardioverted multiple times.

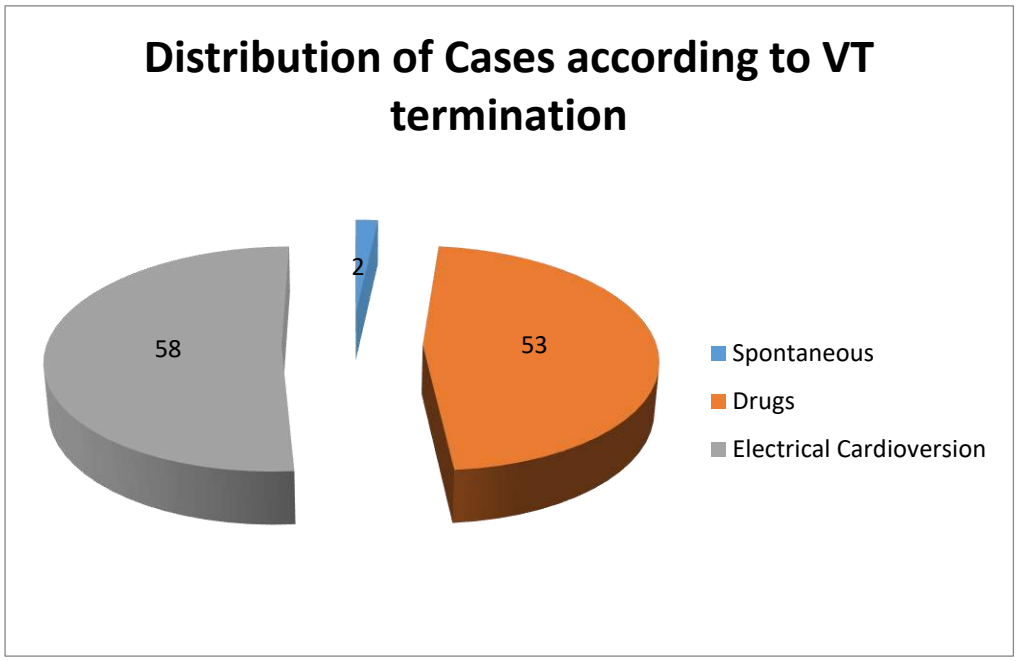
Infusion of lignocaine was given in 18 (15.9%) patients and infusion of amiodarone was given in 21(18.5%) patients.

IABP was inserted in 13 (11.5%) patients.

9 (7.9%) patients were intubated and put on mechanical ventilation.

In cases of refractory VT not responding to medicines, sympathetic ganglion block was done in 6 (5.3) patients.

FIGURE 9: DISTRIBUTION OF CASES ACCORDING TO VT TERMINATION



ELECTROLYTE ABNORMALITIES

TABLE 10: DISTRIBUTION OF PATIENTS ACCORDING TO THE MAGNESIUM LEVELS

Hypomagnesemia	Frequency	Percentage
Present	5	4.4
Absent	108	95.6
TOTAL	113	100.0

Abnormal Levels (hypomagnesemia) are seen in 4.4% patients while normal levels are seen among 95.6% patients.

FIGURE 10: DISTRIBUTION OF PATIENTS ACCORDING TO THE MAGNESIUM LEVELS

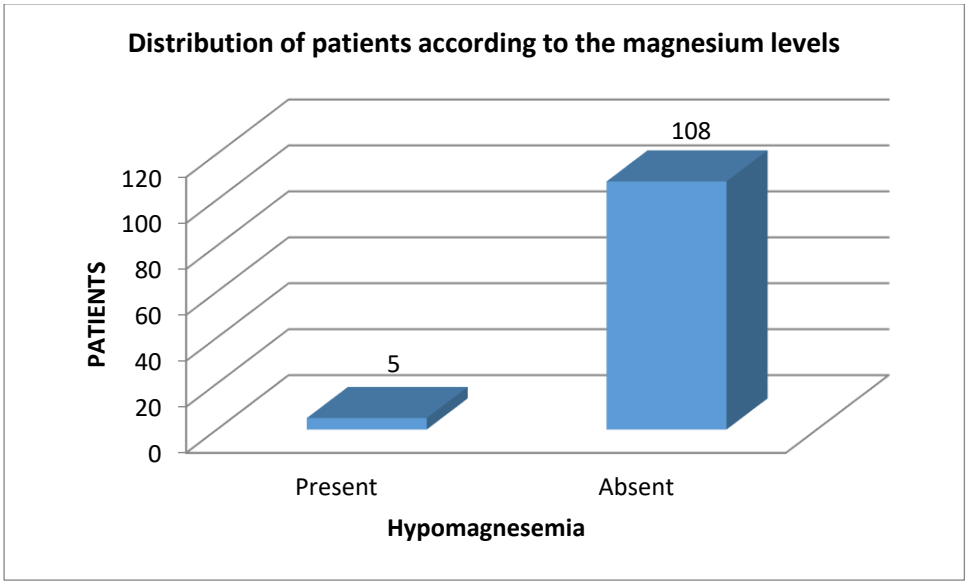
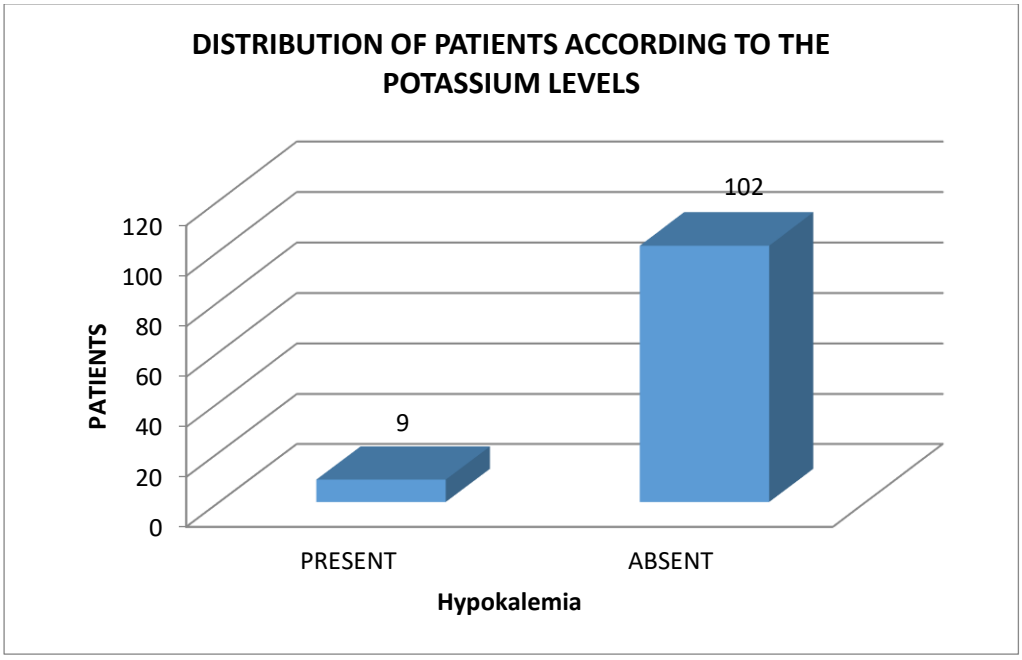


TABLE 11: DISTRIBUTION OF PATIENTS ACCORDING TO THE POTASSIUM LEVELS

Hypokalemia	Frequency	Percentage
PRESENT	9	8.0
ABSENT	102	92
TOTAL	113	100.0

Hypokalemia was seen in 8% of the patients while it was absent in 92% patients in present study.

FIGURE 11: DISTRIBUTION OF PATIENTS ACCORDING TO THE POTASSIUM LEVELS



ECHOCARDIOGRAPHIC FINDINGS

TABLE 12: LVEF OF PATIENTS STUDIED

Echocardiography	Frequency	Percentage
<20	3	2.7
21-30	52	46.0
31-40	36	31.9
41-50	11	9.7
>50	11	9.7
Total	113	100.0

Left ventricular ejection fraction (LVEF) was found >40 in 19.4% patients, in 31.9% patients it was between 31-40, in 46% patients it was 21-30 while in 2.7% it was <20.

FIGURE 12: LVEF OF PATIENTS STUDIED

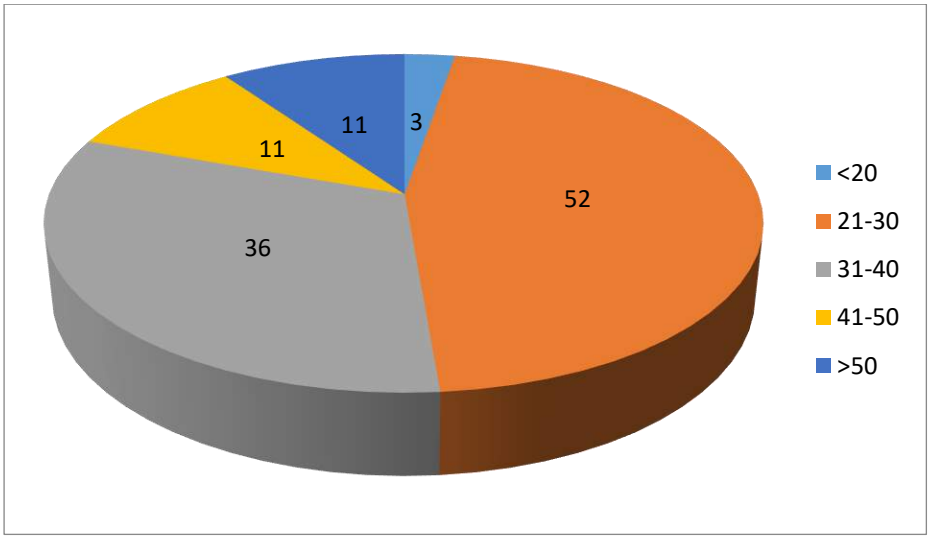


TABLE 13: DISTRIBUTION OF PATIENTS ACCORDING TO CORONARY ANGIOGRAPHY (CAG)

CAG findings	No. Of patients	Percent
Single vessel disease	41	36.4
Triple vessel disease	15	13.3
Double vessel disease	20	17.6
Not done	37	32.7
Total	113	100

In maximum cases single vessel disease was present (36.4%) followed by double vessel disease which was observed in 17.6% patients. 13.3% patients presented with triple vessel disease in CAG. In 32.7% patients CAG was not performed.

FIGURE 13: DISTRIBUTION OF PATIENTS ACCORDING TO CAG FINDINGS

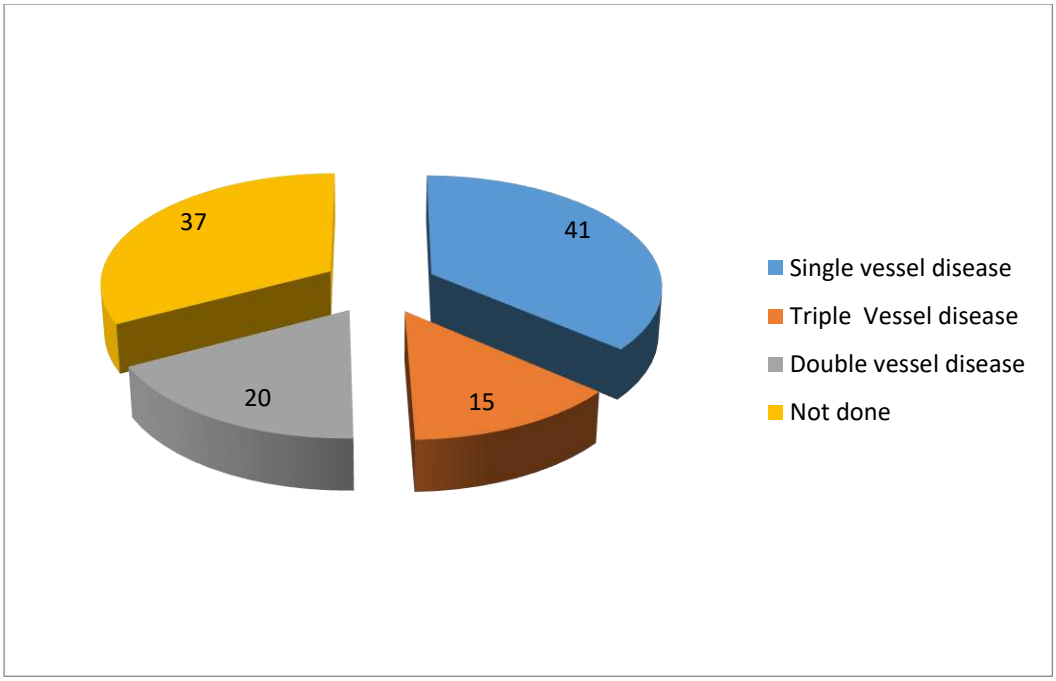


TABLE 14: DISTRIBUTION OF CASES ACCORDING TO INTERVENTION DONE

CAD(61)	Frequency	Percent
PCI	34	30.1
CABG	13	11.5
AICD	14	12.4

30.1% underwent PCI, 11.5% underwent CABG while in 12.4% AICD was done.

FIGURE 14: DISTRIBUTION OF CASES ACCORDING TO INTERVENTION DONE

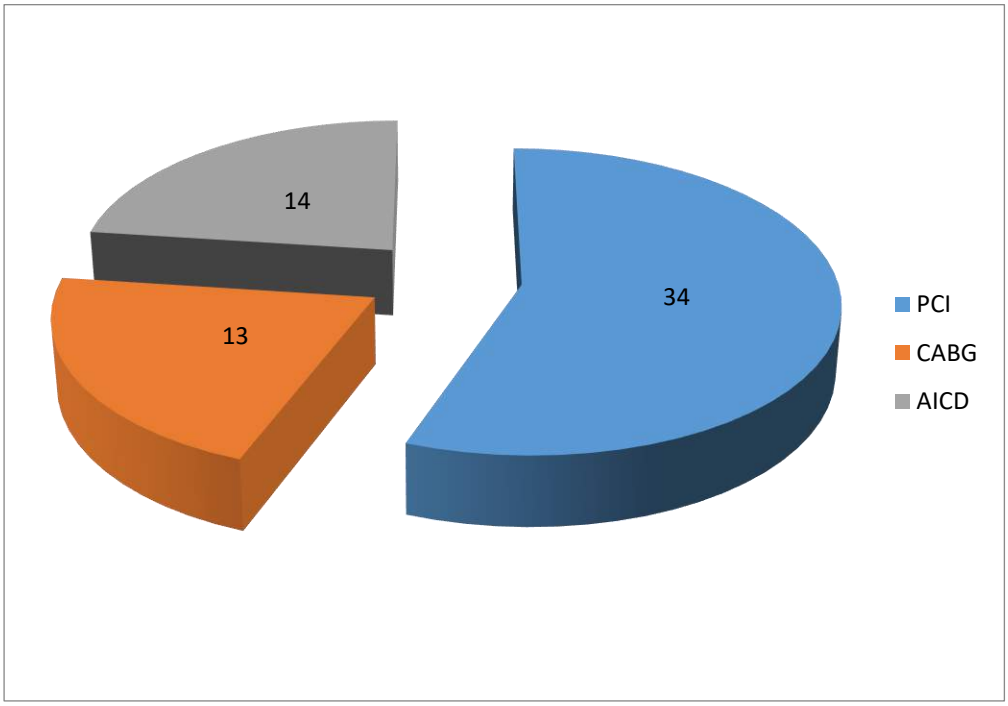


TABLE 15: DISTRIBUTION OF PATIENTS ACCORDING TO THE DRUGS AT DISCHARGE

Drugs at discharge	No. Of patients	Percentage of patients
Beta blocker	75	79.6
Amiodarone	52	46
Mexilitine	38	33.6
Calcium channel blocker	2	1.8

It was observed that maximum patients were on Beta blocker (79.6%) followed by Amiodarone (46%), which was given in patients with recurrent VT and mexilitine (33.6%) was prescribed in patients with drug resistant VT and patients with prolonged QTc interval. Calcium channel blocker was administered in 1.8% patients for additional co morbidities.

FIGURE 15: DISTRIBUTION OF PATIENTS ACCORDING TO THE DRUGS AT DISCHARGE

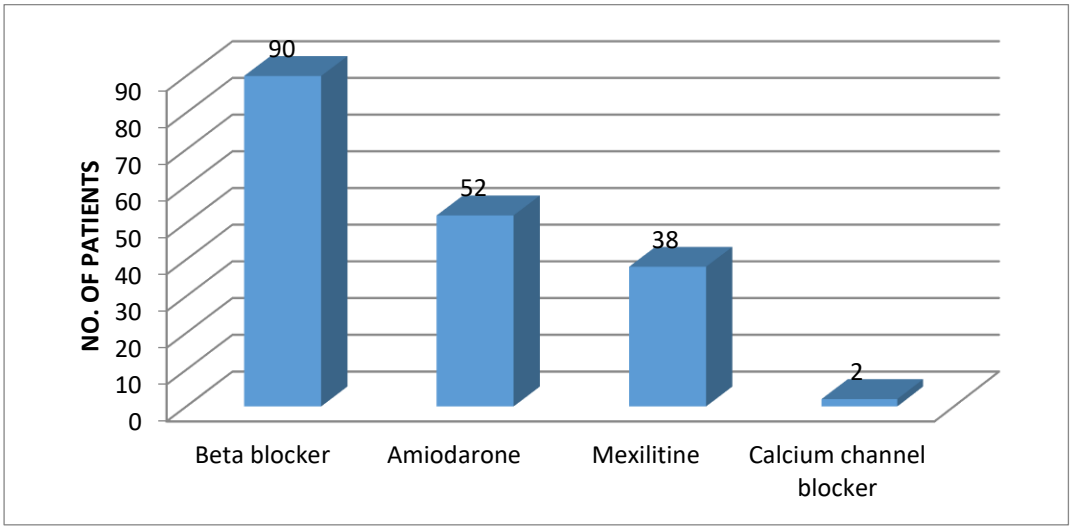


TABLE 16: DISTRIBUTION OF CASES ACCORDING TO IN HOSPITAL OUTCOME

In hospital outcome	No. of patients	percentage of patients
No recurrence	29	25.7
Recurrence	76	67.3
Death	8	7.1
Total	113	100.0

In 25.7% patients the episode of VT did not occur again while in 67.3 % there was recurrence of VT episode while 7.1% patients died.

It was observed that more death rate was seen in patients who were unstable hemodynamically and had LVEF<20. While recurrence was more in patients who were stable and had LVEF between 20-30. Patients with STEMI and NSTEMI also reported with more recurrence and death rate.

FIGURE 16: DISTRIBUTION OF CASES ACCORDING TO IN HOSPITAL OUTCOME

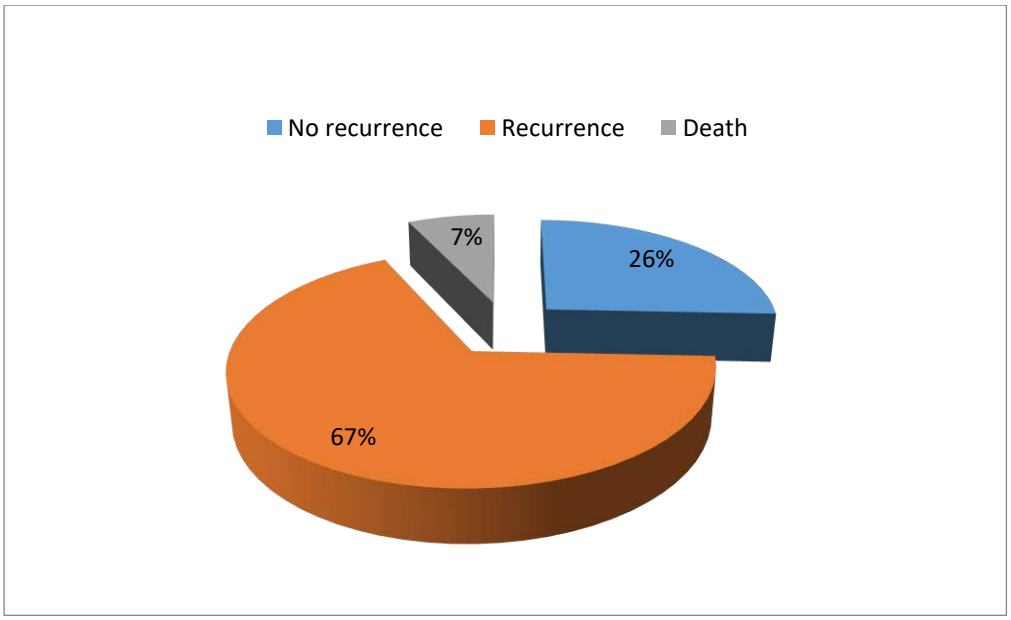


TABLE 17: DISTRIBUTION OF CASES ACCORDING TO 6 MONTHS OUTCOME

6 month outcome	No. of patients	%age of patients
Recurrence	58	51.3
Death	55	48.7
Total	113	100.0

In 51.3% there was reoccurrence of VT episode in six months while 48.7% patients died. Main factor that attributed to recurrence and death was reduced LVEF and presence of CAD.

FIGURE 17: DISTRIBUTION OF CASES ACCORDING TO 6 MONTHS OUTCOME

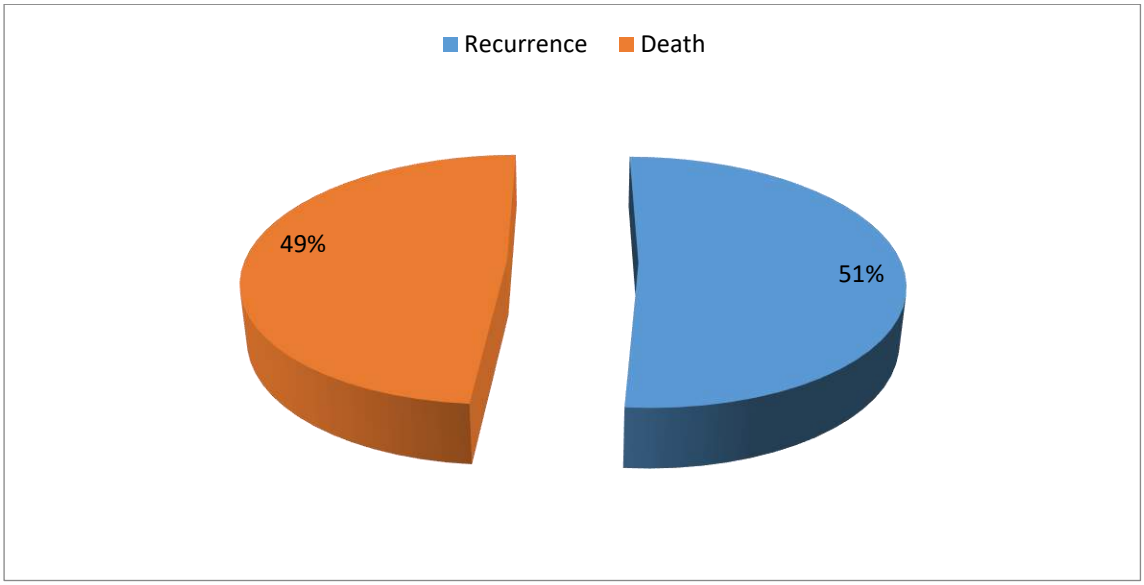


TABLE 18: ASSOCIATION OF LVEF WITH HEMODYNAMIC STABILITY OF THE PATIENT

LVEF	Stable		unstable		Total		P-value
	n	%	n	%	n	%	
<20	0	0.0%	3	6.5%	3	2.7%	<.001
21-30	9	13.4%	43	93.5%	52	46.0%	
31-40	36	53.7%	0	0.0%	36	31.9%	
41-50	11	16.4%	0	0.0%	11	9.7%	
>50	11	16.4%	0	0.0%	11	9.7%	
Total	67	100.0%	46	100.0%	113	100.0%	

It was observed that patients whose LVEF was <20 were all the cases were unstable while patients with LVEF between 21-30, 93.50% were unstable. Patients with LVEF >30 were mostly stable. This association was statistically significant.

FIGURE 18: ASSOCIATION OF LVEF WITH HEMODYNAMIC STABILITY OF THE PATIENT

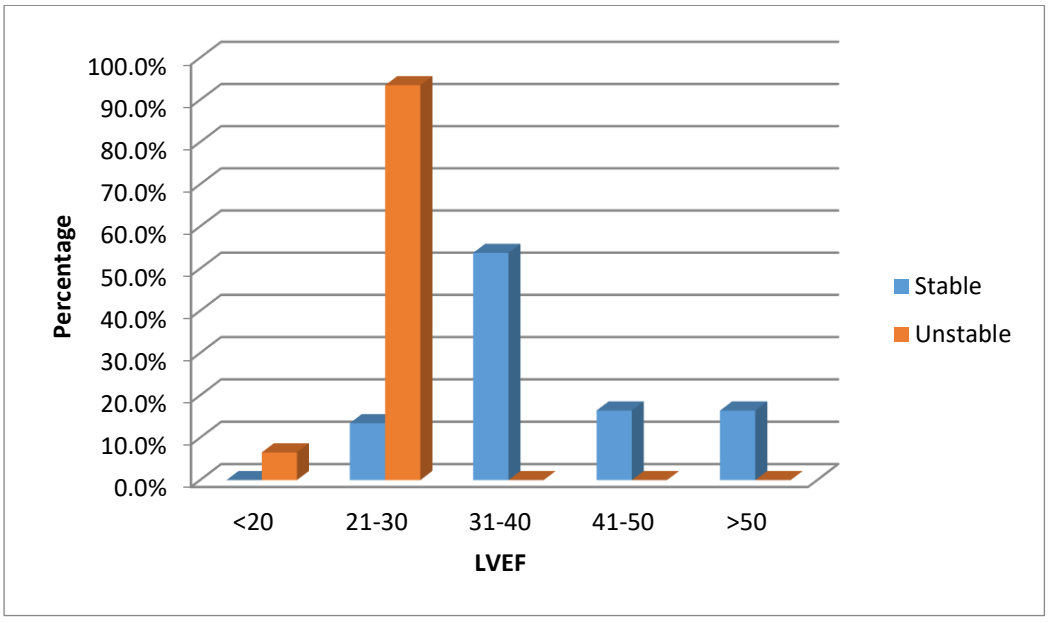


TABLE 19: ASSOCIATION OF 6 MONTHS OUTCOME WITH HEMODYNAMIC STABILITY OF THE PATIENT

6 months outcome	Stable		Unstable		Total		P-value
	n	%	n	%	n	%	
Recurrence	44	65.7%	14	30.4%	58	51.3%	<.001
Death	23	34.3%	32	69.6%	55	48.7%	
Total	67	100.0%	46	100.0%	113	100.0%	

Significant association was seen among 6 month outcome and hemodynamic stability of the patient with unstable patients reporting more deaths than stable patients.

FIGURE 19: ASSOCIATION OF 6 MONTHS OUTCOME WITH HEMODYNAMIC STABILITY OF THE PATIENT

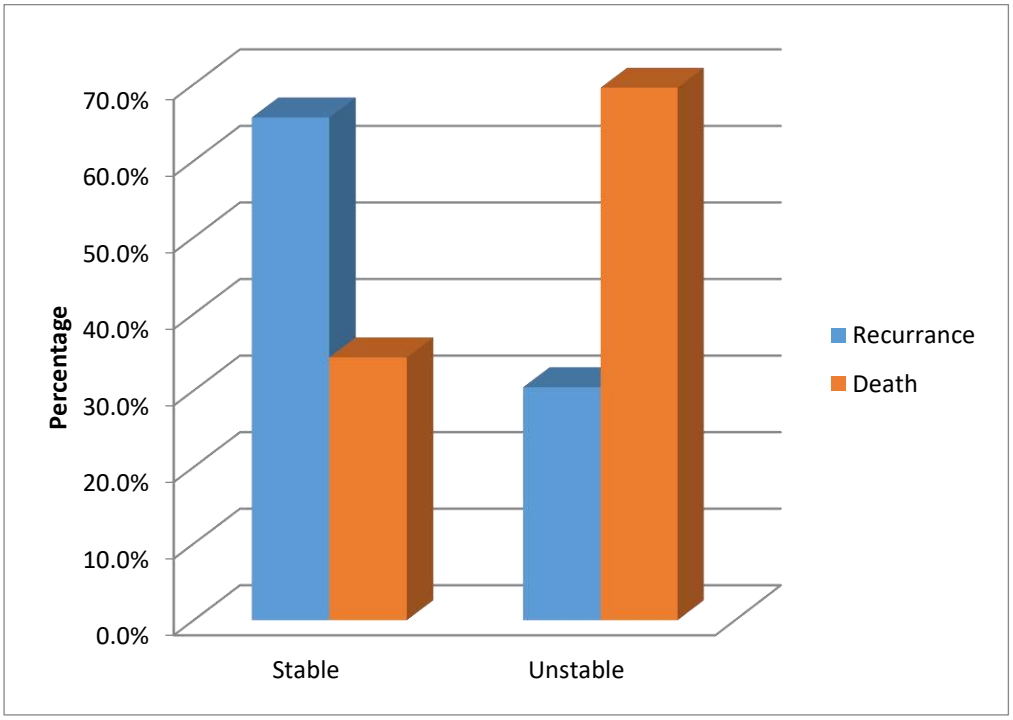


TABLE 20: ASSOCIATION BETWEEN VT MORPHOLOGY AND HEMODYNAMIC STABILITY

	Stable		Unstable		Total		P-value
	n	%	n	%	n	%	
Monomorphic	54	80.60%	17	37.00%	71	62.80%	<.001
Polymorphic	13	19.40%	29	63.00%	42	37.20%	
Total	67	100.00%	46	100.00%	113	100.00%	

Significant association was seen among patient in relation to VT morphology and hemodynamic stability. It was observed patients who were unstable had mostly polymorphic VT.

FIGURE20: ASSOCIATION BETWEEN VT MORPHOLOGY AND HEMODYNAMIC STABILITY

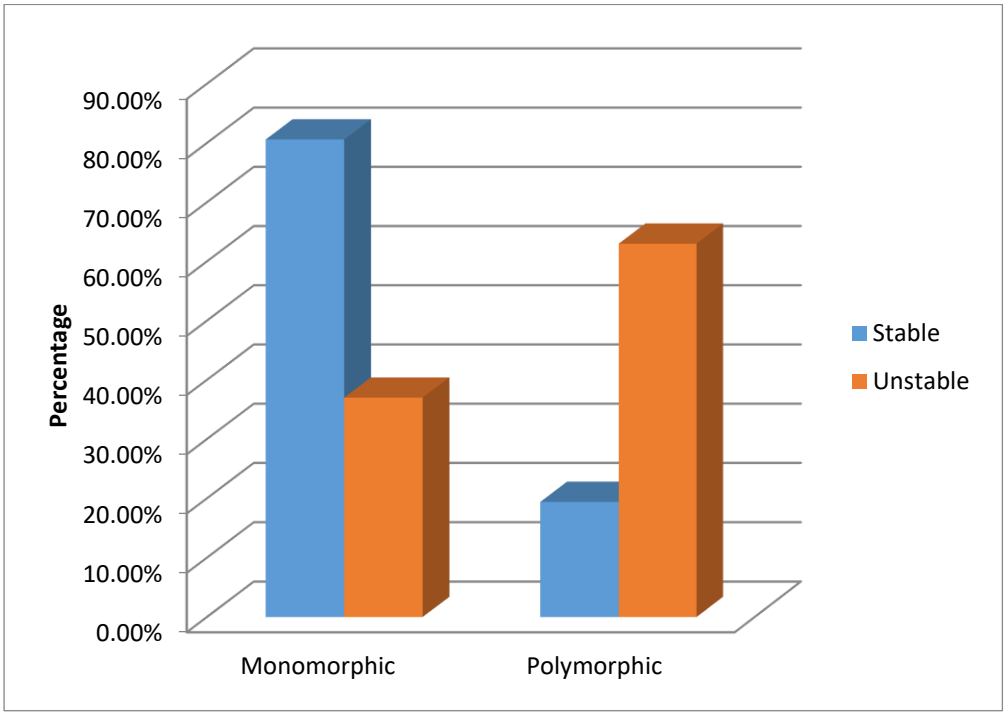


TABLE 21: ASSOCIATION BETWEEN CAUSE OF VT AND HEMODYNAMIC STABILITY

Causes	Stable		Unstable		Total		p-value
	n	%	n	%	n	%	
CAD	32	47.80%	29	63.00%	61	54.00%	.04
Cardiomyopathy	22	32.80%	10	21.70%	32	28.30%	
RHD	5	7.50%	2	4.30%	7	6.20%	
congenital	2	3.00%	5	10.90%	7	6.20%	
Diselectrolytemia	6	9.00%	0	0.00%	6	5.30%	
Total	67	100.00%	46	100.00%	113	100.00%	

It was observed that patients with CAD were significantly more unstable than other causes.

FIGURE 21: ASSOCIATION BETWEEN CAUSE OF VT AND HEMODYNAMIC STABILITY

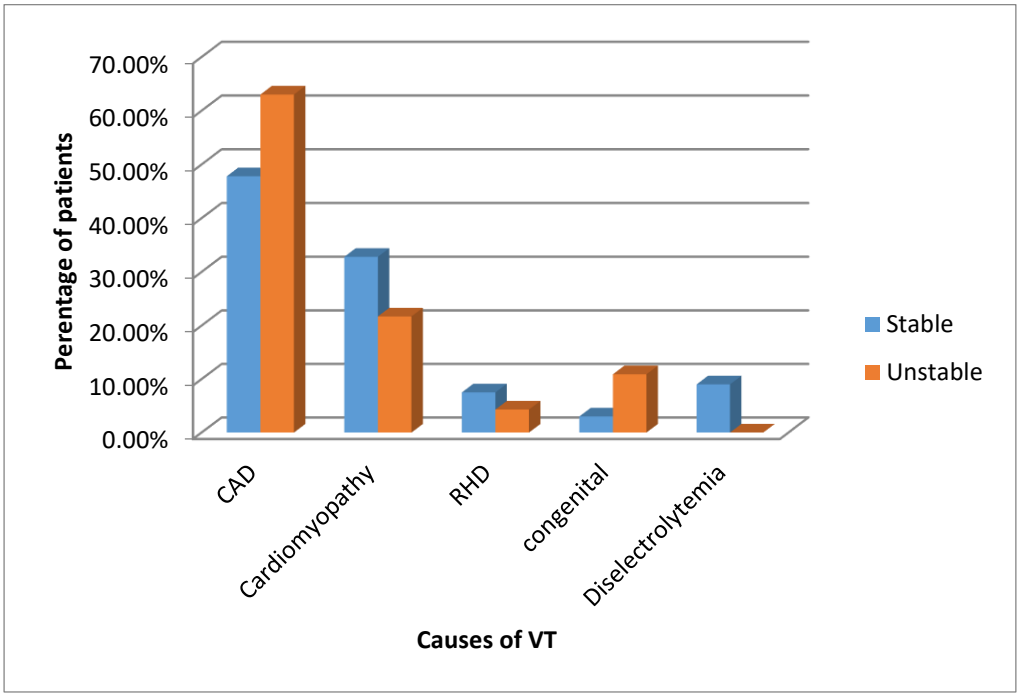


TABLE 22: ASSOCIATION BETWEEN CAUSE OF VT AND 6 MONTH OUTCOME

Causes	Recurrence		Death		Total		p-value
	n	%	n	%	n	%	
CAD	15	25.9%	46	83.6%	61	54.0%	<.001
Cardiomyopathy	26	44.8%	6	10.9%	32	28.3%	
RHD	6	10.3%	1	1.8%	7	6.2%	
congenital	6	10.3%	1	1.8%	7	6.2%	
Diselectrolytemia	5	8.6%	1	1.8%	6	5.3%	
Total	58	100.0%	55	100.0%	113	100.0%	

It was observed that 6 month mortality was significantly more in patients with CAD then other causes.

FIGURE 22: ASSOCIATION BETWEEN CAUSE OF VT AND 6 MONTH OUTCOME

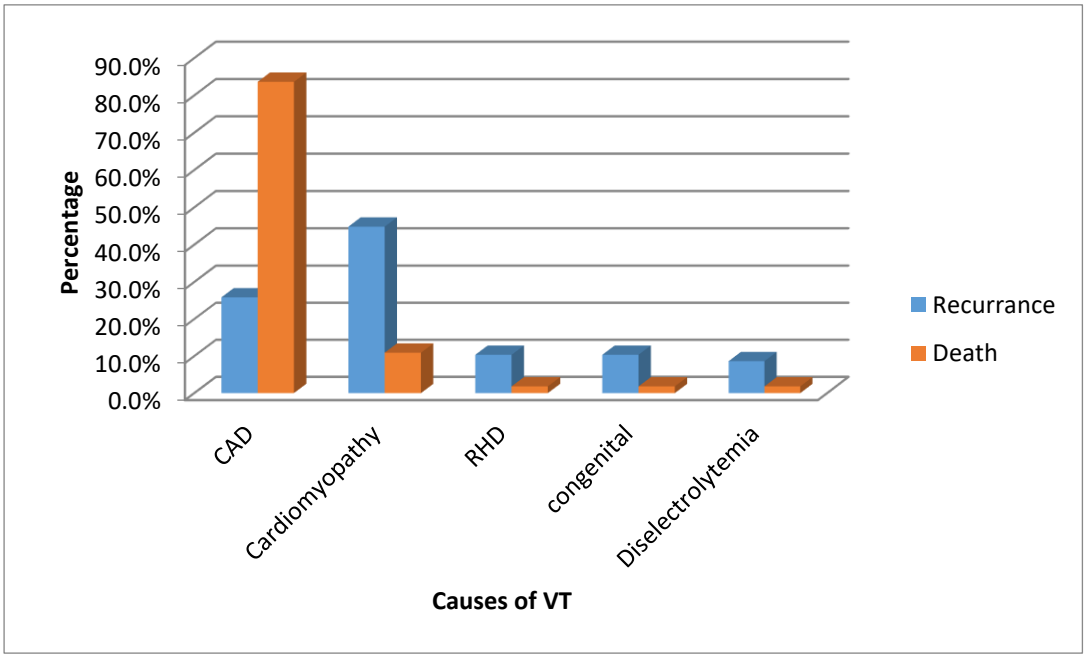


TABLE 23: ASSOCIATION BETWEEN VT MORPHOLOGY AND 6 MONTH MORTALITY

	Recurrence		Death		Total		P-value
	n	%	n	%	n	%	
Monomorphic	46	79.3%	25	45.45%	71	62.8%	<.001
Polymorphic	12	20.7%	30	55.55%	42	37.2%	
Total	58	100%	55	100%	113	100.00%	

It was observed that patients with polymorphic VT morphology had more 6 month death rate than patients with monomorphic VT.

FIGURE 23: ASSOCIATION BETWEEN VT MORPHOLOGY AND 6 MONTH MORTALITY

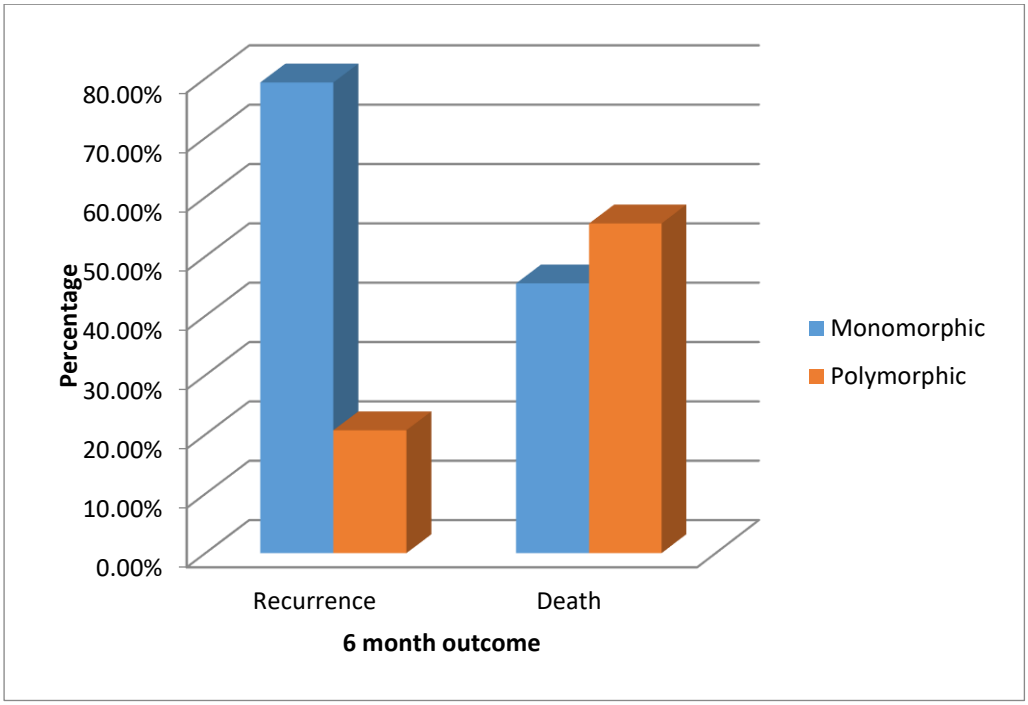


TABLE 24: ASSOCIATION BETWEEN CAUSE OF VT AND VT MORPHOLOGY

Causes	Monomorphic changes		Polymorphic changes		Total		p-value
	n	%	n	%	n	%	
CAD	29	40.80%	32	76.20%	61	54.00%	<.001
Cardiomyopathy	26	36.60%	6	14.30%	32	28.30%	
RHD	5	7.00%	2	4.80%	7	6.20%	
congenital	5	7.00%	2	4.80%	7	6.20%	
Diselectrolytemia	6	8.50%	0	0.00%	6	5.30%	
Total	71	100.00%	42	100.00%	113	100.00%	

It was observed that polymorphic VT were significantly more in patients with CAD then other causes.

FIGURE 24: ASSOCIATION BETWEEN CAUSE OF VT AND VT MORPHOLOGY

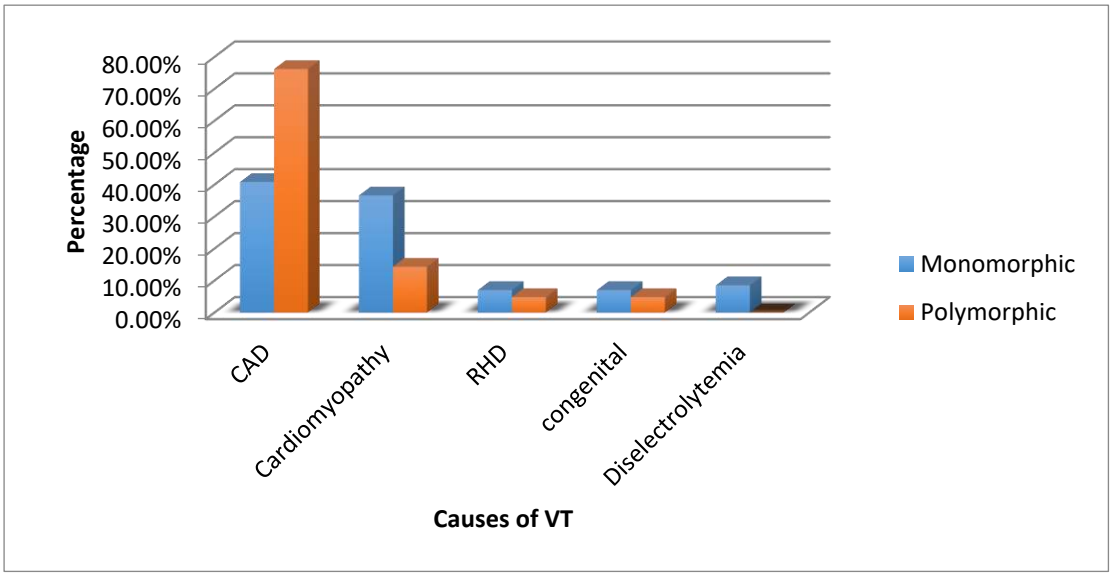
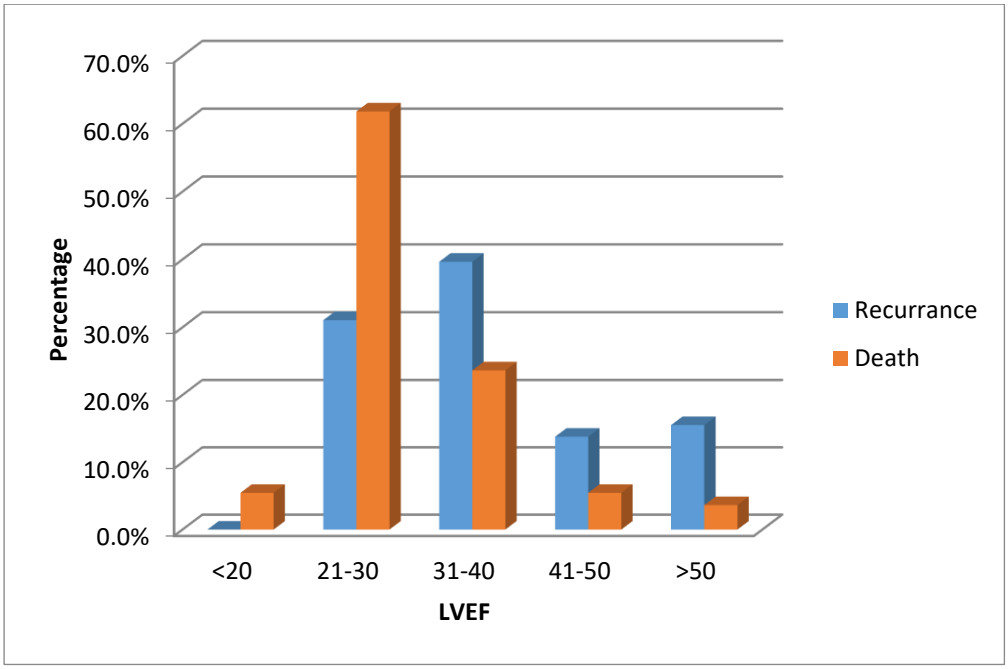


TABLE 25: ASSOCIATION OF LVEF WITH 6 MONTH OUTCOME

LVEF	Recurrence		Death		Total		P-value
	n	%	n	%	n	%	
<20	0	0.0%	3	5.5%	3	2.7%	.002
21-30	18	31.0%	34	61.8%	52	46.0%	
31-40	23	39.7%	13	23.6%	36	31.9%	
41-50	8	13.8%	3	5.5%	11	9.7%	
>50	9	15.5%	2	3.6%	11	9.7%	
Total	58	100.0%	55	100.0%	113	100.0%	

It was observed that patients whose LVEF was <30 had significantly more mortality rate as compared to patients with LVEF more than 30.

FIGURE 25: ASSOCIATION OF LVEF WITH 6 MONTH OUTCOME



Discussion

The present study is a prospective observational study conducted in the cardiology department of DMC hospital. All cases with definite ventricular tachycardia admitted in the hospital or presenting in emergency department were included in the study. The patients were defined into two groups: hemodynamically stable and hemodynamically unstable. Hemodynamic instability is defined as need for immediate intervention due to loss of consciousness, hypotension or occurrence of congestive heart failure. Otherwise patients will be defined as hemodynamically stable.

The results obtained are discussed below:

In our study, out of 113 patients studied 76 patients (67.3%) were males and 37 patients (32.70 %) were females. Maximum patients in both male and female category were in the age group 51-70 years with about 67.2% (76) patients. The results of the present study were in accordance to the study conducted by Sudan et al [62] who also reported the male predominance (55%) in their study however the mean age in their study was 47 years but in present study the mean age was 63.05±11.5 years. Our study was in accordance to the study conducted by Domanovits who observed the 68% males in their study and the median age was median age 63 years. [37]

Existing literature on VT also shows male predominance. Lampert R et al [67] studied gender differences in ventricular arrhythmias in patients with CAD and observed that women were less likely to develop VT/VF episodes.

In present study maximum patients at the time of admission presented with chest pain (58.4%) followed by shortness of breath (41.6%), dizziness (13.3%), palpitations (10.7%), syncope (8%) least no of patients presented with fatigue (1.8%).

Similar results were obtained by Sudan R et al who observed that the most common presenting symptom was chest pain seen in a total of 40% patients. This was in accordance to the present study. [68]

It was observed that maximum patients had risk factors of Diabetes (33.6%), followed by hypertension (23.8%) and Smoking (18.6%).

In the review presented by Foth C et al who reported that major risk factors for ventricular tachycardia are hypertension, previous myocardial infarction and ST-segment changes at presentation. In present study also maximum patients presented with hypertension and diabetes.[69]

CAD was the major cause of VT (54%) followed by cardiomyopathy seen in 28.3% , rheumatic heart disease and congenital cause was seen in 6.2%, 5.3% showed diselectrolytemia.

Krishnan SC et al reported that Patients with coronary artery disease most commonly caused recurrent ventricular tachycardia.[70] Domanovits H et al [37] in their study reported CAD as a major cause for VT with 45% patients followed by acute myocardial infarction (31%); cardiomyopathy (9%); idiopathic VT (7%), arrhythmogenic right ventricular dysplasia (4%). Almas et al in their study reported Myocardial ischemia in 43.5% of the patients, 23.2% had cardiomyopathy, followed by 33.3% miscellaneous. [71]

Koplan et al [72] in their article reported that the most common cause of ventricular fibrillation is acute coronary ischemia, whereas a myocardial scar from prior infarct is the most common cause of sustained monomorphic VT in patients with structural heart disease.

Ischemic heart disease is the most common cause of sustained ventricular arrhythmias. Acute coronary ischemia is a cause of polymorphic VT or ventricular fibrillation (VF) and is probably the most common cause of out-of-hospital sudden death. During acute ischemia, the leakage of potassium leads to increased extracellular potassium that depolarizes myocytes in the ischemic border zone. [73,74] This

depolarization leads to electrical heterogeneity of conduction and refractoriness that provide a substrate for reentry, resulting in polymorphic VT and/or VF.

Among 61 patients of CAD 25/61 were of NSTEMI (40.9%) 26 were of STEMI (42.7%) and 10 cases were of angina (16.4%). Almas et al [71] found out of the patients who had myocardial ischemia (43.5%), 76.6% had non-ST elevation myocardial infarction, 20% patients had ST elevation myocardial infarction and 3.3% had unstable angina. In a study by Sudan et al 13 patients developed sustained VT during the course of their hospital stay in the immediate post MI period. 8 patients had ST elevation MI (STEMI) and the rest 5 patients had Non ST elevation MI (NSTEMI). [68]

Out of 113 patients studied, 46 patients (40.7%) were unstable hemodynamically. 67 (59.3%) of patients were hemodynamically stable. Sudan et al in their study in 2018 observed that total 69% of the patients were hemodynamically stable while 31% were unstable. Almas et al [71] found hemodynamic instability in 50% patients. In another study by Sudan et al in 2018 they observed that total of 23 patients (66%) were hemodynamically stable at the time of VT and 12 patients (34%) were in a state of hemodynamic instability. In a study by Domanovits [37] 77.3% patients were stable while 27.7% were unstable. All these studies were in accordance to the present stud where also most of the patients were in stable condition.

A total of 59.3% of the patients were hemodynamically stable at presentation, underlining the fact that it is misleading to believe that VT always causes severe hemodynamic disturbance [75]. The diagnosis of VT, therefore, should be based on the history and ECG findings, not on clinical status.

Out of 113 patients studied, 71 patients (62.8%) were having monomorphic VT while 42% (37.2%) have polymorphic VT. In a study by Domanovits et al [37] also maximum patients reported monomorphic VT. In a study by Sudan et al also maximum patients developed

patients developed monomorphic VT which was in accordance to the present study. Structural heart disease leading to Sustained monomorphic VT is most commonly caused by re-entry involving a region of myocardial scar. This type of VT occurs in the absence of acute ischemia. [76,77] Ventricular scars leading to reentrant VT also occur in nonischemic cardiomyopathies, including idiopathic dilated cardiomyopathy, hypertrophic cardiomyopathy, infiltrative heart disease (eg, sarcoidosis), right ventricular dysplasia, and after repair of congenital heart disease or valvular heart disease.

In 1.8% cases VT termination was spontaneous, in 51.3% electrical cardioversion was used, drugs were used in 46.9%. Out of all 58 patients who were given electrical cardioversion,15/58 (13.27%) were cardioverted multiple time.

Infusion of IV lignocaine was given 18 (15.9%) patients and infusion of amiodarone was given in 21 (18.5%) patients. IABP was done in 13 (11.5%) patients. 9 7.9%) patients were intubated and put on mechanical ventilation. In cases of refractory VT not responding to medicines, sympathetic ganglion block was done in 6(5.3), patients

In a study by Sudan et al [68] DC cardioversion was given to all the 12 patients with hemodynamic instability. DC cardioversion restored sinus rhythm in all these patients. All the 23 patients who were hemodynamically stable at the time of VT received intravenous drugs initially, which reverted VT in 18 patients (78%). The remaining 5 patients were given DC cardioversion in addition, to revert VT. Domonovits et al [37] (n=75) in their study showed that in the primarily stable patients tachycardia terminated spontaneously in three cases. In 33 out of 55 patients, first-line i.v. antiarrhythmic treatment was successful. In 22 not responding to antiarrhythmic treatment VT was terminated electrically. One of the unstable patients with repetitive torsade de pointes and an intermittent Bradycardia was stabilised by continuous orciprenalin infusion. Immediate cardioversion terminated VT in the remaining 16 unstable patients.

In another study by rahul sudan et al [62] spontaneous termination of sustained VT occurred in seven patients in hemodynamically stable group. Antiarrhythmic drug terminated VT in 53 patients and 14 patients received combined therapy (drug and electrical) after antiarrhythmic drug failure. Out of total 67 patients receiving antiarrhythmic drugs, 33 (49%) received amiodarone, 25 (36%) received lignocaine 9 (13%) received sotalol and 32 patients (48%) received combination therapy including intravenous beta-blocker. One patient in unstable group with polymorphic VT after receiving dc-cardioversion developed repetitive bradycardia related torses de pointes that was managed initially with isoproterenol infusion and subsequently received dual chamber implantable cardioverter defibrillator (ICD) with pacing.

The role of hypokalemia in causing ventricular arrhythmias and VT in general is well recognized. Osadcii Eet al [78] studied the mechanisms of hypokalemia induced ventricular arrhythmogenicity and found that hypokalemia is a common biochemical finding in cardiac patients and often represents a side effect of diuretic therapy or may result from endogenous activation of renin angiotensin system and high adrenergic tone. Hypokalemia is an independent risk factor contributing to reduced survival of cardiac patients and increased incidence of arrhythmic deaths.

Electrolyte abnormalities were present in a significant number of our patients with hypokalemia present in as many as 8% of patients.

Hypomagnesemia as a cause of polymorphic VT is well mentioned in literature. Schinichi et al [79] described polymorphic VT associated with hypomagnesemia in the patients of Sheehan syndrome. Steven R et al[80] have described polymorphic VT associated with hypomagnesemia inpatients of ulcerative colitis and parenteral hyper alimentation in non-digitalized patients. Hypomagnesemia was present in 4.4% patients in present study.

Left ventricular systolic dysfunction represented by reduced ejection fraction secondary to CAD or cardiomyopathy servesas a substrate for genesis of VT. Left ventricular ejection fraction (LVEF)was decreased in 90.3% of our patients which is well supported by the available literature as majority of these patients have either CAD or various types of cardiomyopathies. Roderick Tung et al [81] while working on catheter ablation of VT observed that VT most commonly occurs in the weak end heart musculature.

Left ventricular ejection fraction (LVEF) was found >50 and between 41-50 in 9.7% patients each, in 31.9% patients it was between 31-40, in 46% patients it was 21-30, while in 2.7% it was <20.

It was observed that patients whose LVEF was <20 were all the cases were unstable while patients with LVEF between 21-30, 93.50% were unstable. Patients with LVEF >30 were mostly stable. This association was statically significant. In present study significant difference was obtained in the ejection fraction of hemodynamically stable and unstable patients. Mean value of LVEF in stable patients was 38.61±9.9 while in unstable patients it was 23.93±3.3 .the results were obtained by Sudan et al [62] were in discordance to the present study where LVEF was 50.6 ±14.5 while for 48.6 ±14.5. and no significant difference between both. In present study no significant change was seen in hypokalemia and hypomagnesia in stable and unstable patients.

In maximum cases single vessel disease was present (36.4%) followed by double vessel disease which was observed in 17.6% patients. 13.3% patients presented with triple vessel disease in CAG. In 32.7% patients CAG was not performed.

In a study by Sudan et al also CAG was done in 28 patients (80%). Most common lesion seen was double vessel disease (DVD) in 13 patients (46%). [62]

Asymptomatic patients with non-sustained ventricular tachycardia (VT) and no underlying cardiac co-morbidities require no additional therapy.

Patients that are symptomatic and with cardiac co-morbidities should be started on a beta-blocker due to favorable efficacy and safety profile. [82,83] The nondihydropyridine calcium channel blockers are contraindicated in the setting of structural heart disease or heart failure with reduced ejection fraction [84] except in special cases such as left fascicular VT.

Patients with sustained monomorphic ventricular tachycardia (SMVT) that are unstable should be managed following advanced cardiac life support (ACLS) guidelines. Hemodynamically stable patients should be pharmacologically cardioverted using an anti-arrhythmic medication. Intravenous amiodarone or procainamide can be used for this purpose. Procainamide will terminate between 50% and 80% of ventricular tachycardias, and it will slow the conduction of those that it does not terminate. [85,86] Amiodarone will convert about 30% of patients to sinus rhythm but is very effective in reducing the reversion rate of refractory SMVT. [57, 87-90]

The first-line therapy for chronic treatment of patients with ischemic heart disease and VT is a beta-blocker therapy which is associated with a reduced risk of sudden cardiac death. In patients with ischemic heart disease who have recurrent VT despite beta-blocker therapy, amiodarone or sotalol can be initiated however neither therapy has been associated with a decrease in mortality. [91-93] Amiodarone plus a beta-blocker has been associated with a greater decrease in the number of ICD shocks compared to sotalol monotherapy. [88-93]

Patients with ischemic heart disease that survive sudden cardiac arrest due to ventricular tachycardia, or experience hemodynamically unstable or stable sustained ventricular tachycardia, should have an implantable cardiac defibrillator (ICD) placed if their estimated meaningful survival is greater than 1 year. [90,94]

Cardioversion [95-97]

If a patient with VT is hemodynamically unstable, immediate cardioversion is necessary. This is accomplished by a 100-200J

biphasic shock. All reversible factors must be corrected. VT in patients with an MI should be treated with lidocaine. If the patient has torsades de pointes then magnesium sulfate is preferred.

It was observed that maximum patients were prescribed at discharged beta blockers (79.6%) followed by amiodarone (46%), mexilitine (33.6%) and calcium channel blocker (1.8%). Mexilitine (33.6%) was prescribed in patients with drug resistant VT and patients with prolonged QTc interval.

In 22.1% patients the episode of VT did not occur again while in 69% there was reoccurrence of VT episode while 8.8% patients died. It was observed that more death rate was seen in patients who were unstable hemodynamically and had LVEF<20. While recurrence was more in patients who were stable and had LVEF between 20-30. Patients with STEMI and NSTEMI also reported with more recurrence and death rate.

Similar results were observed by Domanovits et al [37] who also noted more death in unstable patients than stable with the significant difference (p<.05).Sudan et al [68] also reported same results with significant association between death and hemodynamic stability thus hemodynamic stability being important predictor of mortality. In a study by Almas et al [71] Ventricular tachycardia was associated with a significant in hospital mortality of 13.7%.

Significant association was seen among patient in relation to VT morphology and hemodynamic stability. It was observed patients who were unstable had mostly polymorphic VT. Domanovits et al [37] studies a clinical profile of VT presenting in emergency department and showed that only ECG characteristics that correlated with the hemodynamic instability was the polymorphic pattern. This was in accordance with the present study.

It was observed that 6 month mortality was significantly more in patients with CAD then other causes. In 51.3% there was reoccurrence of VT episode while 48.7% patients died. The 6 month mortality was

relatively high in present study. This could be due to COVID 19 pandemic present during the study. Peterson et al in 2021 conducted a study and reported that Patients with CAD and COVID-19 have higher rates of comorbidities, inpatient mortality and need for renal replacement therapy compared to their non-CAD counterparts. However, CAD in itself was not associated with mortality after adjusting for other covariates, suggesting that other factors may play a larger role in the increased mortality and poor outcomes in these patients. [98] Loffi et a in 2019 reported Patients with COVID-19 and CAD have an exceedingly higher risk of mortality, which is mainly attributable to the burden of comorbidities rather than to a direct effect of CAD per se. [99]

In present study however the cause of the higher mortality could not be ascertained but it could be attributed to COVID 19 pandemic.

Summary and Conclusion

- In our study, out of 113 patients maximum patients were in age group 51-70 years. The mean age of the patients was 63.5. There were 76 males and 37 females.
- CAD was the most common cause of VT (54%) followed by cardiomyopathy (28.3%). Other less common causes were rheumatic heart disease, congenital heart disease and diselectrolytemia.
- Associated risk factor of Diabetes mellitus was found in 33.6% patients followed by hypertension (23.8%) and Smoking (18.6%).
- Out of 113 patients studied, 46 patients (40.7%) were hemodynamically unstable. 67 (59.3%) of patients were hemodynamically stable.
- 71 patients (63%) were having monomorphic VT while 42 patients (37%) have polymorphic VT. In 9.7% VT occurred at the time of admission. In 57.6% cases VT occurred within 24 hours of admission while in 32.7% cases it occurred after 24 hours.
- In 1.8% cases VT termination was spontaneous, in 51.3% electrical cardioversion was used, drugs were used in 46.9 %.
- Hypomagnesemia and Hypokalemia was seen in 4.4% and 8% patients
- Left ventricular ejection fraction (LVEF) was found >40 in 19.4% patients, in 31.9% patients it was between 31-40, in 46% patients it was 21-30 while in 2.7% it was <20.
- In maximum cases single vessel disease was present (36.4%). 30.1% underwent PCI, 11.5% underwent CABG while in 12.4% AICD was done.

- It was observed that maximum patients were on Beta blocker (79.6%) followed by amiodarone (46%), which was given in patients with recurrent VT and mexilitine (33.6%) was prescribed in patients with drug resistant VT and patients with prolonged QTc interval.
- During hospital stay, in 25.7% patients the episode of VT did not occur again while in 67.3 % there was recurrence of VT episode while 7.1% patients died.
- It was observed that more death rate was seen in patients who were unstable hemodynamically and had LVEF <30.
- In 51.3% there was reoccurrence of VT episode in six months while 48.7% patients died. Main factor that attributed to recurrence and death was reduced LVEF and presence of CAD.
- More death was observed in patients who were unstable , had LVEF <30, had CAD and in patients with polymorphic VT morphology.

Conclusion

Ventricular tachycardia is an important cause of sudden death. CAD remains the leading cause of VT. Hemodynamically unstable VT occurs more frequently in polymorphic VT. Anti-arrhythmic drugs are used to terminate VT but most effective method of VT termination is DC cardioversion.CAD, Hemodynamic instability and reduced ejection fraction contribute to overall mortality in VT.

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				chief complaint						Risk Factors/comorbidities						drugs given			Lab investigation								
SNO.	MRD NO.	AGE	SEX	Chest pain	Shortness of breath	Palpitations	Syncope	DIZZINESS	FATIGUE	smoking	THYROID	diabetes	hypertension	CVA	tb/ba	BETA BLOCKER	AMIODARONE	MEXILITINE	HB	UREA	CREATININE	SODIUM	CALCIUM	ALBUMIN	SGPT	SGOT	TROP I	BNP
1	937973	59	M	YES	YES	-	-	-	-	YES	-	YES	-	-	-	YES	-	-	12.7	116	1.2	134	7.8	3.4	76	262	postive	731
2	937411	56	M	YES	YES	-	-	-	-	-	-	YES	-	-	-	YES	-	-	13.8	25	0.6	136	8.8	3.3	32	200	postive	1210
3	938385	63	F	YES	-	YES	-	-	-	-	-	-	YES	-	-	-	-	-	12.5	63	1.5	131	9.7	3.6	109	174	postive	730
4	932456	70	M	YES	-	-	YES	-	-	-	-	-	-	-	-	YES	YES	-	11.5	272	8.7	135	7.8	2.6	37	20	postive	700
5	534596	63	F	-	YES	YES	-	-	-	-	-	-	YES	-	-	YES	YES	-	10.6	24	0.7	141	8	2.4	70	40	postive	363
6	100345	67	F	-	YES	-	-	-	-	-	-	-	YES	-	-	YES	YES	YES	11.5	64	1.7	14	7.8	3	1474	1321	postive	1360
7	235678	69	M	YES	-	-	-	-	-	-	-	YES	-	-	-	-	YES	YES	12.4	65	1.5	138	7.9	3.6	47	36	postive	730
8	945489	75	M	YES	-	-	-	-	-	-	-	-	-	-	-	YES	-	-	12	28	1.1	132	4.1	3.3	71	41	Negative	60
9	148820	49	M	-	YES	-	-	-	-	-	-	-	-	-	-	YES	-	YES	13.7	14	0.8	141	8	2.7	43	62	postive	443
10	970252	82	M	YES	-	-	-	-	-	YES	-	-	-	-	-	YES	YES	-	12	56	10.1	138	7.8	4.2	28	140	Negative	65
11	1153879	66	M	-	-	-	YES	-	-	-	-	YES	YES	-	-	YES	-	-	12.3	77	1.7	143	3	3	19	33	postive	730
12	168868	65	F	YES	YES	-	-	-	-	-	-	YES	-	-	-	YES	-	YES	9.2	170	2	142	8	3	55	262	postive	740
13	884108	56	F	-	YES	-	-	-	-	-	-	YES	YES	-	-	YES	-	YES	9.4	211	5.6	125	6.7	2.6	10	15	postive	370
14	899170	66	M	YES	YES	-	-	-	-	-	-	-	-	-	-	YES	-	YES	16.7	78	2.1	134	7.9	3	119	92	postive	340
15	234587	47	F	-	-	YES	-	-	-	-	-	-	-	-	-	YES	YES	-	10	27	0.5	140	9.1	4	78	66	postive	1300
16	470267	65	M	YES	-	-	-	-	-	-	-	YES	YES	-	-	YES	YES	-	11.4	27	1	139	4.4	4	19	24	Negative	255
17	1136450	78	M	YES	YES	-	-	-	-	-	-	-	YES	-	-	YES	YES	-	10.4	109	1.6	127	8.6	4	1381	1099	postive	2100
18	352476	76	M	-	YES	-	-	-	-	YES	-	-	-	-	-	YES	-	-	10.8	50	1.3	135	3.8	3.8	28	46	postive	183
19	1126631	65	M	YES	-	-	-	-	-	-	-	-	-	yes	-	YES	YES	-	11.2	34	0.8	134	7.8	3	35	33	postive	255
20	1127732	52	M	YES	-	-	-	-	-	-	-	YES	YES	-	-	-	YES	YES	13.7	30	0.7	140	8.9	4	162	971	postive	370
21	641810	67	M	YES	-	-	-	-	-	-	-	-	-	-	YES	-	YES	-	14.1	34	0.9	135	4.1	3	42	40	postive	360
22	225151	52	M	-	YES	-	-	-	-	-	-	-	-	-	-	YES	-	-	7.4	33	9.8	135	8.5	2.8	19	14	postive	650
23	866570	52	M	-	YES	-	-	-	-	-	-	-	YES	-	-	YES	-	-	10.2	132	2.8	138	8.5	3.3	42	40	postive	750
24	1154047	65	F	YES	-	YES	-	-	-	-	-	-	-	-	-	YES	-	-	10.2	124	1.2	128	8	3	55	262	postive	590
25	1195172	72	M	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	-	12.5	61	1.4	136	5.6	4	66	25	postive	1150
26	876049	73	M	-	-	YES	-	-	-	-	-	-	YES	-	-	YES	YES	-	12.2	20	1	133	8	4.1	18	20	postive	5000
27	947995	60	F	YES	-	-	-	-	-	-	-	-	-	-	-	-	YES	-	11.5	51	1.3	137	8.2	4	53	282	postive	790
28	158280	76	M	YES	-	-	-	-	-	-	-	-	YES	-	-	YES	-	-	11.5	49	1.5	137	7	4	40	140	Negative	2590
29	1031961	59	M	YES	-	-	-	-	-	YES	-	-	YES	-	-	-	YES	-	14.3	27	1.1	136	7.9	3.3	66	80	postive	4798
30	1024118	62	M	YES	-	-	-	-	-	-	-	YES	-	-	-	-	-	YES	14.5	25	1.4	143	7.9	3.2	296	1022	Negative	255
31	958932	70	M	YES	-	-	-	-	-	YES	-	YES	-	-	-	YES	-	YES	9.8	43	1.9	132	7.9	3.1	18	16	postive	1150
32	997018	75	F	YES	-	-	-	-	-	-	-	YES	-	-	-	YES	YES	-	11.3	81	5.3	137	8.1	3.7	9	14	Negative	796
33	995638	63	M	-	YES	-	-	-	-	-	-	-	-	-	-	YES	-	-	12	61	1.3	122	7	3	24	60	postive	1500
34	990221	68	M	YES	YES	-	-	-	-	-	-	YES	-	-	-	YES	YES	YES	15.1	42	0.9	137	8	3	68	96	postive	1130
35	997122	52	F	YES	-	-	-	-	-	-	-	-	-	-	-	-	-	YES	11.6	14	0.3	141	7.4	2.7	34	24	Negative	255
36	989705	61	M	-	YES	-	-	-	-	-	-	YES	-	-	-	YES	-	YES	16.1	93	4.1	134	10	3.9	2327	1408	postive	1010
37	989659	40	M	-	-	-	YES	-	-	-	-	-	-	-	-	YES	YES	YES	12.1	36	0.9	130	8.8	3.5	280	129	Negative	255
38	372597	67	F	-	-	-	-	YES	-	-	-	-	-	-	-	YES	-	-	12.7	24	0.6	134	8	2.9	39	74	postive	536
39	1010956	90	M	-	-	-	-	YES	-	-	-	YES	-	-	-	YES	YES	-	9.3	176	10.7	126	8.4	3.8	25	35	postive	570
40	342780	70	F	-	YES	-	-	-	-	-	-	-	-	-	-	YES	YES	-	9	180	10	120	8	3	20	30	postive	968
41	926541	70	M	-	-	-	-	YES	-	-	-	-	-	-	-	YES	-	-	12.8	35	0.8	135	10.6	3.5	43	57	postive	100
42	1008646	63	F	YES	-	-	-	-	-	-	-	-	-	-	-	YES	-	YES	16.9	34	0.4	139	7	2.7	86	364	postive	195
43	240369	70	M	YES	-	-	-	-	-	YES	-	-	-	-	-	YES	YES	-	11	85	2.3	121	9.3	3.6	15	46	postive	3340
44	1007812	84	M	-	-	-	-	YES	-	-	YES	-	-	-	-	YES	YES	-	13.4	69	2	139	8.2	4.1	22	37	postive	929
45	455280	74	M	YES	-	-	-	-	-	-	-	YES	-	-	-	YES	-	-	8.6	160	3	143	7.7	2.4	15	35	postive	2130
46	202981	65	M	YES	-	-	-	-	-	-	-	YES	-	-	-	YES	-	-	14.8	209	4.5	128	7	2	12	27	postive	930
47	990332	24	M	-	YES	-	-	-	-	-	-	YES	-	-	-	-	YES	-	13.6	69	2.2	132	8	3.5	707	288	postive	570
48	990348	58	M	YES	-	-	-	-	-	-	-	-	-	-	-	YES	-	-	11.2	31	101	133	9.5	4.1	24	39	postive	82
49	10088510	65	F	YES	-	-	-	-	-	-	-	YES	YES	-	-	YES	-	YES	11.1	49	0.5	137	9	4	94	168	postive	435
50	631638	60	F	-	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	8.9	131	2.9	124	8	4	11	27	postive	690
51	1006493	82	F	YES	-	-	-	-	-	-	YES	-	-	-	-	YES	-	YES	12	33	0.6	139	9.2	3.8	26	35	postive	235
52	1008972	87	F	YES	-	-	-	-	-	-	-	-	-	-	-	YES	-	-	11	81	5.3	130	7	4.3	28	34	postive	460
53	1007784	82	M	YES	-	-	-	-	-	-	-	-	-	-	-	YES	-	YES	13.1	53	0.9	138	9	4	23	89	postive	128
54	1007812	84	M	-	-	-	-	YES	-	YES	-	YES	YES	-	-	YES	YES	-	13.4	69	2	139	8.2	4.1	22	37	postive	929
55	997142	34	M	-	YES	-	-	YES	-	-	-	-	-	-	-	YES	-	-	15.8	27	0.7	139	8	4	108	574	postive	657
56	985467	63	F	YES	YES	-	-	-	-	-	-	YES	-	-	-	-	YES	YES	10.5	98	2.7	132	9	3.7	25	14	postive	5000
57	575192	77	M	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	-	10.5	54	1.8	135	8.3	3.6	46	33	Negative	885
58	1011723	66	M	-	YES	-	-	-	-	-	-	-	-	-	-	YES	-	-	9	172	2.8	133	10	3.2	166	54	postive	780
59	1115097	58	M	YES	-	-	-	-	-	YES	-	YES	-	-	-	YES	-	YES	11.4	64	1.1	134	7.8	2.8	305	303	postive	870

						Echo findings								others								outcome	
SNO.	MRD NO.	MAGNESIUM	mag cat	POTASSIUM	potassium cat	LVEF%	LVEF% (cat)	IVST (D)	LVID (D)	LVPW(D)	IVST (S)	LVID (S)	LVPW (s)	causes of VT	intervention	CAG	STEMI	ECG	Haemodynamically	types of VT	VT termination	in hosp outcome	6 month outcome
1	937973	2.7	2	5.8	3	25	2	8	51	8	9	41	12	CAD	PCI	Triple vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	death	death
2	937411	2.6	2	4.2	2	32	3	9	46	9	10	38	11	CAD	PCI	double vessel disease	NSTEMI	poly	stable	<24hr	drugs	recurrance	death
3	938385	2.9	3	3.9	2	35	3	11	43	9	11	36	10	CAD	PCI	singe vessel disease	NSTEMI	mono	stable	<24hr	drugs	recurrance	death
4	932456	2.8	2	6.8	3	28	2	6	53	8	8	44	11	CAD	CABG	Triple vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	recurrance	death
5	534596	2.8	2	4.3	2	55	5	10	43	12	12	33	12	Cardiomyopathy	not done	singe vessel disease	NR	poly	stable	adm	drugs	no recurrance	recurrance
6	100345	2.8	2	5	2	32	3	10	52	11	11	40	13	Cardiomyopathy	not done	not done	NR	poly	stable	<24hr	drugs	no recurrance	recurrance
7	235678	2.8	2	4.8	2	28	2	7	39	10	8	33	13	CAD	CABG	singe vessel disease	NSTEMI	mono	stable	adm	drugs	no recurrance	death
8	945489	1.9	2	4.1	2	36	3	9	59	8	11	49	9	CAD	PCI	double vessel disease	NSTEMI	mono	stable	<24hr	drugs	recurrance	death
9	148820	1.7	2	3.6	2	40	3	12	62	12	13	50	13	CAD	PCI	singe vessel disease	NSTEMI	mono	stable	<24hr	drugs	no recurrance	death
10	970252	2.1	2	3.9	2	38	3	8	40	9	9	33	10	Cardiomyopathy	not done	singe vessel disease	NR	mono	stable	>24 hr	E.cardioversion	recurrance	death
11	1153879	2	2	3	1	25	2	8	50	6	10	33	12	CAD	AICD	Triple vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	recurrance	death
12	168868	2	2	3.1	1	20	2	10	51	7	11	39	13	CAD	PCI	Triple vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	death	death
13	884108	2.4	2	3.7	2	36	3	8	50	10	11	41	11	CAD	PCI	singe vessel disease	NSTEMI	mono	stable	<24hr	drugs	recurrance	death
14	899170	2.7	2	6.3	3	18	1	7	47	11	8	43	12	CAD	PCI	Triple vessel disease	STEMI	poly	unstable	<24hr	E.cardioversion	death	death
15	234587	2.5	2	4.4	2	42	4	12	51	13	14	40	14	CAD	PCI	double vessel disease	NSTEMI	mono	stable	>24 hr	E.cardioversion	no recurrance	death
16	470267	2	2	4.3	2	25	2	10	68	11	11	61	13	CAD	AICD	Triple vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	no recurrance	death
17	1136450	2.2	2	5.5	3	32	3	7	58	7	8	48	8	Cardiomyopathy	not done	not done	NR	poly	stable	adm	drugs	no recurrance	recurrance
18	352476	2.7	2	3.8	2	35	3	8	51	8	9	40	9	CAD	PCI	singe vessel disease	UA	poly	stable	<24hr	drugs	no recurrance	recurrance
19	1126631	2.8	2	3.8	2	25	2	8	55	8	9	42	12	Cardiomyopathy	not done	not done	NR	mono	unstable	adm	E.cardioversion	no recurrance	recurrance
20	1127732	2.4	2	4.5	2	27	2	8	59	8	9	47	11	CAD	CABG	double vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	no recurrance	death
21	641810	2.7	2	4.1	2	35	3	12	59	12	14	49	14	RHD	not done	singe vessel disease	NR	mono	stable	>24 hr	E.cardioversion	recurrance	recurrance
22	225151	2.4	2	7.1	3	48	4	16	44	14	18	34	16	CAD	PCI	singe vessel disease	NSTEMI	mono	stable	<24hr	drugs	no recurrance	recurrance
23	866570	2.5	2	6.3	3	20	2	22	51	11	11	42	10	CAD	AICD	Triple vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	recurrance	death
24	1154047	2.1	2	3.6	2	60	5	12	52	12	11	35	12	CAD	PCI	double vessel disease	NSTEMI	mono	stable	<24hr	drugs	no recurrance	recurrance
25	1195172	2	2	5.6	3	34	3	7	58	7	38	48	8	Cardiomyopathy	not done	not done	NR	mono	stable	<24hr	drugs	no recurrance	recurrance
26	876049	2	2	4.8	2	28	2	5	71	6	6	62	9	CAD	CABG	double vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	recurrance	recurrance
27	947995	2.7	2	4.8	2	26	2	6	63	10	7	55	11	CAD	CABG	double vessel disease	STEMI	poly	unstable	<24hr	E.cardioversion	recurrance	death
28	158280	2.9	3	4.2	2	22	2	6	68	5	7	61	8	CAD	AICD	double vessel disease	NSTEMI	mono	unstable	>24 hr	E.cardioversion	no recurrance	recurrance
29	1031961	2.3	2	4.4	2	30	3	10	56	10	12	49	12	RHD	not done	not done	NR	mono	stable	>24 hr	E.cardioversion	no recurrance	recurrance
30	1024118	1.8	2	3.4	1	26	2	8	57	10	9	52	13	Cardiomyopathy	not done	not done	NR	mono	unstable	>24 hr	E.cardioversion	recurrance	death
31	958932	2	2	4.1	2	34	3	8	44	9	9	42	10	Cardiomyopathy	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	recurrance
32	997018	2.4	2	4.2	2	32	3	9	50	5	12	42	6	CAD	PCI	double vessel disease	NSTEMI	mono	stable	<24hr	drugs	recurrance	death
33	995638	2.3	2	4.6	2	40	3	13	44	12	14	35	13	CAD	PCI	Triple vessel disease	STEMI	mono	stable	>24 hr	E.cardioversion	recurrance	death
34	990221	2	2	4.3	2	25	2	6	54	10	8	42	12	CAD	PCI	double vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	death	death
35	997122	2.7	2	4.2	2	60	5	9	44	10	10	28	11	RHD	not done	singe vessel disease	NR	poly	stable	<24hr	drugs	recurrance	recurrance
36	989705	2.1	2	5.4	3	60	5	13	60	13	15	42	15	CAD	PCI	singe vessel disease	NSTEMI	poly	stable	<24hr	drugs	recurrance	recurrance
37	989659	2.5	2	5.6	3	34	3	9	57	9	10	48	13	Cardiomyopathy	not done	not done	NR	mono	stable	>24 hr	E.cardioversion	no recurrance	recurrance
38	372597	2	2	4.8	2	60	5	9	46	9	10	26	10	CAD	PCI	singe vessel disease	NSTEMI	mono	stable	>24 hr	E.cardioversion	recurrance	death
39	1010956	1.8	2	9.4	3	52	5	20	31	10	27	24	11	CAD	PCI	singe vessel disease	NSTEMI	mono	stable	<24hr	drugs	no recurrance	recurrance
40	342780	2	2	4.6	2	50	4	19	30	9	26	22	10	RHD	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	recurrance
41	926541	2.2	2	6.5	3	36	3	8	49	7	9	39	8	Cardiomyopathy	not done	not done	NR	mono	stable	>24 hr	E.cardioversion	recurrance	recurrance
42	1008646	1.89	2	3	1	35	3	7	45	6	8	40	7	Cardiomyopathy	not done	not done	NR	mono	stable	>24 hr	E.cardioversion	recurrance	recurrance
43	240369	2.8	2	9.3	3	22	2	10	71	10	11	62	11	RHD	not done	not done	NR	mono	unstable	>24 hr	E.cardioversion	recurrance	death
44	1007812	1.7	2	5.7	3	26	2	7	63	7	8	55	10	Cardiomyopathy	not done	not done	NR	mono	unstable	<24hr	E.cardioversion	recurrance	recurrance
45	455280	2.4	2	3.4	1	30	3	11	52	10	13	44	12	CAD	PCI	singe vessel disease	UA	mono	stable	<24hr	drugs	recurrance	death
46	202981	2	2	3.7	2	28	2	8	9	11	9	55	12	Cardiomyopathy	not done	not done	NR	mono	stable	>24 hr	E.cardioversion	recurrance	recurrance
47	990332	2	2	5.7	3	18	1	8	61	9	9	58	10	CAD	AICD	Triple vessel disease	STEMI	poly	unstable	<24hr	E.cardioversion	recurrance	death
48	990348	1.9	2	5.4	3	40	3	8	54	8	10	42	11	CAD	PCI	singe vessel disease	UA	poly	stable	<24hr	drugs	recurrance	death
49	10088510	2.6	2	3.9	2	32	3	8	48	9	9	39	10	RHD	not done	singe vessel disease	NR	poly	stable	adm	drugs	recurrance	recurrance
50	631638	2.7	2	5.8	3	30	3	8	46	8	8	40	10	Cardiomyopathy	not done	singe vessel disease	NR	poly	stable	<24hr	drugs	recurrance	recurrance
51	1006493	1.96	2	3.8	2	35	3	8	55	9	9	40	10	CAD	PCI	singe vessel disease	UA	poly	stable	<24hr	drugs	recurrance	death
52	1008972	2	2	4	2	25	2	12	51	16	13	49	10	RHD	not done	not done	NR	mono	unstable	>24 hr	E.cardioversion	no recurrance	recurrance
53	1007784	2.7	2	5.2	3	30	3	8	63	7	9	50	10	Cardiomyopathy	not done	not done	NR	mono	stable	>24 hr	E.cardioversion	no recurrance	recurrance
54	1007812	1.7	2	5.7	3	26	2	7	63	7	8	55	10	CHD	not done	not done	NR	mono	unstable	>24 hr	E.cardioversion	recurrance	recurrance
55	997142	2	2	4.5	2	22	2	6	54	10	7	49	12	Cardiomyopathy	not done	not done	NR	mono	unstable	<24hr	E.cardioversion	recurrance	recurrance
56	985467	2.5	2	5.1	3	30	3	8	44	9	8	49	10	CAD	PCI	singe vessel disease	UA	mono	stable	>24 hr	E.cardioversion	no recurrance	death
57	575192	1.6	1	5.1	3	30	3	4	61	10	5	52	12	Cardiomyopathy	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	recurrance
58	1011723	2	2	6.1	3	22	2	9	64	10	10	56	11	CAD	AICD	double vessel disease	STEMI	poly	unstable	<24hr	drugs	no recurrance	death
59	1115097	2.1	2	3.8	2	28	2	6	52	9	7	45	10	CHD	not done	singe vessel disease	NR	poly	unstable	adm	E.cardioversion	no recurrance	death

				chief complaint						Risk Factors/comorbidities						drugs given			Lab investigation								
SNO.	MRD NO.	AGE	SEX	Chest pain	Shortness of breath	Palpitations	Syncope	DIZZINESS	FATIGUE	smoking	THYROID	diabetes	hypertension	CVA	tb/ba	BETA BLOCKER	AMIODARONE	MEXILITINE	HB	UREA	CREATININE	SODIUM	CALCIUM	ALBUMIN	SGPT	SGOT	TROP I	BNP
60	33638	60	M	YES	-	-	-	-	-	-	-	-	-	-	-	YES	-	-	12.5	37	1.8	141	9	3.5	71	43	postive	970
61	992186	52	M	-	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	12.4	59	7.4	137	9	3	44	25	postive	758
62	986749	76	F	-	-	-	-	YES	-	-	-	YES	-	-	-	YES	-	-	10.2	27	1	133	8.3	3	17	31	postive	221
63	899170	60	M	YES	-	-	-	-	-	YES	-	YES	-	-	-	YES	-	-	11.5	35	0.6	134	7.8	2.9	19	105	Negative	16
64	102683	50	F	-	YES	YES	-	-	-	-	-	-	-	-	-	YES	-	-	12.5	42	1.5	133	7	3	1262	16.8	postive	1850
65	171465	61	M	-	YES	YES	-	-	-	YES	-	YES	-	-	-	-	YES	YES	12.9	68	1.2	132	9.6	3.22	555	268	postive	509
66	151510	58	M	YES	-	YES	-	-	-	YES	-	YES	-	-	-	YES	-	YES	13.7	34	0.7	138	7	4	22	37	postive	780
67	912167	61	F	-	YES	-	-	-	-	-	YES	-	YES	-	-	YES	YES	-	9.8	110	3.1	147	8.3	3.2	25	37	postive	3070
68	208489	54	F	-	-	-	-	YES	-	-	-	-	-	-	-	YES	YES	-	11.9	31	1.2	135	9.1	3.3	336	221	postive	2220
69	870542	54	F	YES	YES	-	-	-	-	-	-	-	YES	-	-	YES	-	-	11.4	45	1.1	140	7.9	2.8	14	29	postive	267
70	926541	65	M	-	YES	-	-	-	-	-	-	YES	-	-	-	YES	YES	-	11.6	72	1.3	146	9.2	3	24	28	postive	529
71	890499	70	M	-	-	-	-	YES	-	-	-	-	YES	-	-	YES	-	YES	10.6	51	1.1	144	7	2.9	98	47	postive	1560
72	927172	61	M	-	YES	YES	-	-	-	YES	-	-	YES	-	-	YES	-	-	15.2	23	0.6	134	9	3	22	48	postive	501
73	927486	59	M	YES	-	-	-	-	-	-	-	-	-	-	-	-	YES	YES	15.2	259	12.4	139	8.7	3.73	568	59	postive	1820
74	133310	73	M	-	YES	-	YES	-	-	YES	-	-	YES	-	-	YES	-	-	9.9	59	1.4	141	4.8	4.3	17	23	postive	496
75	931580	48	M	-	YES	-	-	-	-	-	YES	-	-	-	-	YES	-	-	11	48	1.1	132	8	4	44	65	postive	2200
76	932456	70	M	YES	-	-	YES	YES	-	-	-	YES	-	-	-	YES	-	-	13.3	33	0.9	143	9	4	70	88	postive	315
77	933815	53	F	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	-	12.4	51	1.3	138	7.7	3.3	17	22	Negative	559
78	939723	70	F	YES	YES	-	YES	YES	-	-	-	-	YES	-	-	YES	YES	-	13.5	28	0.8	133	9.5	4.3	13	16	Negative	1040
79	937087	45	M	YES	-	-	-	-	-	YES	-	-	-	-	-	YES	YES	-	10	15	0.6	136	9	4.2	26	88	postive	325
80	936054	78	F	YES	-	-	-	-	-	-	-	-	YES	-	-	YES	-	-	11.1	41	0.9	132	9	4	14	16	postive	349
81	936761	65	F	-	YES	-	-	-	-	-	-	YES	-	-	-	YES	-	YES	10.1	92	1.8	135	8.8	4	14	14	postive	1330
82	1143878	63	F	YES	-	-	-	-	-	-	-	YES	-	-	-	YES	YES	-	11.8	40	1.1	125	8	4	36	31	postive	958
83	939654	60	M	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	-	12.4	25	1	129	8.5	3.8	24	22	postive	1200
84	945202	73	M	YES	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	12.1	35	2.1	138	8	4.2	85	134	Negative	73
85	9944966	57	F	-	YES	-	YES	-	-	-	-	YES	-	-	-	YES	YES	-	11.1	30	0.6	142	10.3	3.8	50	57	Negative	936
86	364834	59	M	YES	YES	-	-	-	-	-	-	YES	-	-	-	YES	-	-	12.7	116	1.2	134	7.8	3.4	76	262	postive	731
87	1056534	56	F	YES	YES	-	-	-	-	-	-	-	-	-	-	YES	-	-	11.8	76	2.1	130	7	4	174	164	postive	5000
88	1013519	70	M	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	-	12,5	47	1.1	142	8.9	3.9	15	15	postive	230
89	101956	70	M	-	YES	-	-	-	YES	YES	-	-	-	-	-	YES	YES	-	9.3	176	10.7	126	8.4	3.8	25	35	postive	440
90	928344	63	M	-	-	-	-	-	YES	-	-	YES	YES	-	-	YES	-	YES	12	61	1.1	133	8	2.8	42	22	postive	1610
91	149412	74	M	YES	-	-	-	-	-	YES	-	-	-	-	-	YES	-	-	12.2	58	1.4	137	8	2	44	28	postive	598
92	965822	48	M	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	-	13.8	31	1	143	8.6	4.1	109	287	Negative	25
93	962212	67	M	-	-	-	YES	-	-	-	-	-	-	-	-	-	-	YES	14.9	35	0.7	139	8.1	3.7	44	38	Negative	142
94	9577222	54	F	-	YES	-	-	YES	5	-	-	-	-	-	-	YES	-	-	10.2	121	2.2	134	7.8	2.9	1420	546	postive	649
95	939702	62	F	-	-	-	-	YES	-	-	-	YES	YES	-	-	-	-	YES	7.5	72	0.9	133	8	3.2	1871	860	postive	1870
96	387890	71	F	-	YES	-	-	-	-	-	-	-	YES	-	-	YES	-	YES	9	26	2.4	140	9.4	3.4	10	14	postive	5000
97	1133496	54	M	-	YES	-	-	YES	-	-	-	YES	YES	-	-	YES	-	YES	12.4	25	0.4	137	7.6	2.8	43	32	postive	4800
98	606683	40	M	-	-	YES	YES	-	-	-	-	-	-	-	-	YES	YES	-	14.6	23	0.6	137	7	3.6	32	85	postive	90
99	1103191	46	M	YES	-	YES	-	-	-	YES	-	-	-	-	-	-	YES	-	12	33	1.8	140	9	4	24	22	postive	1200
100	953357	49	F	YES	-	-	-	YES	-	-	-	-	YES	-	-	-	YES	-	9.6	19	0.5	136	8.3	4.1	18	23	Negative	604
101	697666	56	M	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	YES	12	34	2	140	9	4	25	22	postive	5000
102	164506	59	M	YES	YES	-	-	-	-	-	-	-	-	-	-	-	-	YES	12.9	35	0.7	137	9	4	80	455	Negative	188
103	235683	50	F	YES	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	12.5	42	1.5	133	8.6	4	1260	1608	postive	509
104	975372	58	F	-	-	YES	-	-	-	-	-	YES	-	-	-	YES	-	YES	13.7	34	0.7	134	7	3.3	25	37	postive	3070
105	7294635	59	M	YES	YES	-	-	-	-	YES	-	-	YES	-	-	YES	-	-	11.4	45	1.1	140	7.9	2.8	14	29	postive	267
106	930472	59	M	YES	-	-	-	-	-	YES	-	-	-	-	-	-	YES	YES	15.2	250	12.4	139	8.7	3.74	568	59	postive	1820
107	112569	60	M	YES	-	-	-	-	-	-	-	-	-	-	-	YES	YES	-	12.4	25	1	129	8.5	3.8	24	22	postive	1200
108	883956	59	M	YES	YES	-	-	-	-	YES	-	-	-	-	-	YES	-	-	12.7	116	1.2	134	7.8	3.4	76	262	postive	731
109	883592	67	M	-	YES	-	-	-	-	-	YES	-	-	-	-	YES	-	-	11.5	64	1.7	14	8	4	1474	1321	postive	1360
110	995634	42	M	YES	-	-	-	-	-	-	-	YES	-	-	-	YES	YES	-	13.7	45	0.8	140	9.3	3.2	26	27	postive	1400
111	1019611	64	M	YES	YES	-	-	-	-	YES	-	YES	-	-	-	-	YES	YES	14.1	68	1.5	145	7.8	3	65	62	postive	543
112	1013647	55	M	YES	-	-	-	-	-	-	-	YES	-	-	-	YES	-	-	11.7	35	1.54	140	8.8	3.6	11	26	postive	560
113	1016344	70	M	-	YES	-	-	-	-	-	-	-	-	-	YES	YES	-	-	11	33	2	134	9	3	60	59	postive	570

						Echo findings								others								outcome	
SNO.	MRD NO.	MAGNESIUM	mag cat	POTASSIUM	potassium cat	LVEF%	LVEF% (cat)	IVST (D)	LVID (D)	LVPW(D)	IVST (S)	LVID (S)	LVPW (s)	causes of VT	intervention	CAG	STEMI	ECG	Haemodynamically	types of VT	VT termination	in hosp outcome	6 month outcome
60	33638	2.6	2	4.9	2	45	4	9	49	10	10	32	11	CAD	PCI	singe vessel disease	NSTEMI	poly	stable	<24hr	drugs	no recurrance	death
61	992186	2	2	3.6	2	25	2	9	60	9	10	40	10	Cardiomyopathy	not done	not done	NR	mono	unstable	>24 hr	E.cardioversion	recurrance	recurrance
62	986749	2.8	2	3.8	2	35	3	7	43	11	8	35	12	CAD	PCI	double vessel disease	NSTEMI	mono	stable	<24hr	drugs	recurrance	recurrance
63	899170	2.4	2	4	2	60	5	11	46	12	12	34	13	CAD	PCI	singe vessel disease	NSTEMI	mono	stable	<24hr	drugs	recurrance	recurrance
64	102683	2	2	3.7	2	22	2	22	54	10	11	45	11	Cardiomyopathy	not done	singe vessel disease	NR	mono	unstable	<24hr	drugs	recurrance	recurrance
65	171465	2.49	2	4.1	2	28	2	9	60	10	10	52	11	CAD	CABG	singe vessel disease	UA	mono	stable	<24hr	drugs	recurrance	death
66	151510	1.7	2	3.5	2	25	2	6	63	7	7	56	10	CHD	not done	not done	NR	mono	unstable	<24hr	E.cardioversion	recurrance	recurrance
67	912167	2	2	4.2	2	28	2	9	48	9	10	38	12	CAD	CABG	double vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	recurrance	recurrance
68	208489	1.6	1	4.5	2	45	4	8	44	12	10	32	12	Cardiomyopathy	not done	singe vessel disease	NR	poly	stable	<24hr	drugs	no recurrance	recurrance
69	870542	1.8	2	3.6	2	20	2	9	69	9	10	59	10	CAD	AICD	singe vessel disease	UA	poly	unstable	>24 hr	E.cardioversion	recurrance	death
70	926541	2.9	3	4.2	2	24	2	9	66	10	10	59	11	Cardiomyopathy	not done	singe vessel disease	NR	poly	unstable	>24 hr	E.cardioversion	recurrance	death
71	890499	2.3	2	4.4	2	24	2	5	72	10	7	66	11	CHD	not done	singe vessel disease	NR	poly	unstable	adm	E.cardioversion	recurrance	recurrance
72	927172	2.7	2	4.1	2	28	2	8	56	9	9	9	11	CAD	CABG	singe vessel disease	NSTEMI	mono	stable	<24hr	drugs	recurrance	recurrance
73	927486	2.5	2	4.1	2	44	4	9	49	11	10	32	11	Cardiomyopathy	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	recurrance
74	133310	2.6	2	4.8	2	46	4	12	72	12	13	65	13	CAD	PCI	double vessel disease	NSTEMI	mono	stable	<24hr	drugs	no recurrance	death
75	931580	2.5	2	4.3	2	30	3	15	60	15	17	50	17	Cardiomyopathy	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	recurrance
76	932456	2.8	2	5	2	28	2	8	53	8	6	44	11	CAD	CABG	double vessel disease	NSTEMI	mono	stable	<24hr	drugs	recurrance	recurrance
77	933815	2.1	2	4.1	2	40	3	9	49	10	10	41	11	Cardiomyopathy	not done	singe vessel disease	NR	mono	stable	<24hr	drugs	recurrance	recurrance
78	939723	2	2	4.5	2	25	2	9	62	11	10	54	12	CAD	AICD	Triple vessel disease	STEMI	poly	unstable	<24hr	E.cardioversion	recurrance	death
79	937087	2	2	3.4	1	25	2	8	44	10	10	44	11	CAD	AICD	double vessel disease	STEMI	poly	unstable	<24hr	drugs	recurrance	death
80	936054	2.8	2	4	2	60	5	12	48	12	48	33	13	Cardiomyopathy	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	recurrance
81	936761	2.2	2	4.7	2	55	5	12	48	11	10	44	11	CAD	PCI	singe vessel disease	UA	mono	stable	<24hr	spontaneous	no recurrance	death
82	1143878	2.8	2	4.6	2	26	2	5	39	8	6	34	9	CAD	PCI	singe vessel disease	UA	mono	unstable	<24hr	E.cardioversion	recurrance	death
83	939654	2.2	2	4.5	2	25	2	3	61	9	4	53	8	Cardiomyopathy	not done	not done	NR	mono	unstable	>24 hr	E.cardioversion	recurrance	death
84	945202	2.5	2	3.5	2	20	2	9	54	9	10	40	10	CHD	not done	singe vessel disease	NR	mono	unstable	<24hr	E.cardioversion	recurrance	recurrance
85	9944966	2.5	2	4	2	25	2	11	85	10	12	78	11	CAD	AICD	double vessel disease	STEMI	poly	unstable	<24hr	E.cardioversion	recurrance	death
86	364834	2.7	2	5.8	3	25	2	8	51	8	9	41	12	CAD	PCI	Triple vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	death	death
87	1056534	2.8	2	4.4	2	25	2	4	53	10	5	46	12	CAD	AICD	double vessel disease	STEMI	poly	unstable	>24 hr	drugs	recurrance	death
88	1013519	0.4	1	4.1	2	35	3	8	64	9	9	53	11	Diselectrolytemia	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	recurrance
89	101956	1.8	2	9.4	3	52	5	20	24	10	31	24	11	CHD	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	recurrance
90	928344	2	2	5.1	3	28	2	11	48	7	12	38	8	CAD	CABG	singe vessel disease	NSTEMI	mono	stable	<24hr	drugs	recurrance	death
91	149412	2.8	2	4.3	2	30	3	7	57	10	8	42	14	CHD	not done	singe vessel disease	NR	mono	stable	<24hr	drugs	recurrance	recurrance
92	965822	2	2	3.1	1	28	2	8	50	10	9	44	12	Diselectrolytemia	not done	not done	NR	mono	stable	<24hr	E.cardioversion	recurrance	recurrance
93	962212	1.3	1	4	2	33	3	11	52	10	9	40	12	Diselectrolytemia	not done	not done	NR	mono	stable	adm	drugs	no recurrance	recurrance
94	9577222	1.7	2	3.7	2	42	4	6	47	10	7	38	11	CAD	PCI	singe vessel disease	NSTEMI	mono	stable	<24hr	drugs	recurrance	recurrance
95	939702	1.9	2	4	2	44	4	8	48	8	9	24	9	CAD	PCI	singe vessel disease	NSTEMI	mono	stable	<24hr	drugs	no recurrance	recurrance
96	387890	2	2	3.1	1	32	3	13	51	12	15	35	14	Diselectrolytemia	not done	singe vessel disease	NR	mono	stable	adm	E.cardioversion	recurrance	recurrance
97	1133496	1.6	1	3.5	2	28	2	8	63	9	9	54	10	Diselectrolytemia	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	death
98	606683	2	2	3.6	2	30	3	10	59	5	14	49	6	Cardiomyopathy	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	recurrance
99	1103191	2	2	4.2	2	52	5	12	45	12	13	30	13	Cardiomyopathy	not done	singe vessel disease	NR	mono	stable	<24hr	E.cardioversion	recurrance	recurrance
100	953357	2	2	4.1	2	20	2	8	59	8	9	53	9	CAD	AICD	not done	UA	mono	unstable	<24hr	E.cardioversion	recurrance	death
101	697666	2	2	4	2	14	1	6	64	7	8	59	8	CAD	PCI	Triple vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	death	death
102	164506	2.5	2	4	2	22	2	7	58	7	58	48	8	Cardiomyopathy	not done	not done	NR	mono	unstable	adm	E.cardioversion	recurrance	death
103	235683	2.5	2	4	2	28	2	9	60	10	10	52	11	Cardiomyopathy	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	death
104	975372	1.7	2	3.5	2	45	4	8	44	12	10	32	11	CAD	PCI	singe vessel disease	NSTEMI	mono	stable	<24hr	spontaneous	no recurrance	recurrance
105	7294635	1.8	2	3.6	2	20	2	9	69	9	10	59	10	CAD	AICD	Triple vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	recurrance	death
106	930472	2.5	2	4.1	2	44	4	9	49	11	10	32	11	Cardiomyopathy	not done	not done	NR	mono	stable	<24hr	drugs	recurrance	recurrance
107	112569	2.2	2	4.5	2	25	2	3	61	9	4	53	8	Cardiomyopathy	not done	not done	NR	mono	unstable	<24hr	E.cardioversion	recurrance	recurrance
108	883956	2.7	2	5.6	3	25	2	8	51	8	9	41	12	CAD	AICD	double vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	recurrance	death
109	883592	2.5	2	5	2	32	3	10	52	11	11	40	13	CAD	CABG	double vessel disease	NSTEMI	mono	stable	<24hr	E.cardioversion	death	death
110	995634	2	2	4.9	2	28	2	11	57	11	12	50	12	CAD	PCI	singe vessel disease	STEMI	poly	unstable	>24 hr	E.cardioversion	recurrance	death
111	1019611	2.7	2	3	1	34	3	12	61	13	13	49	14	Diselectrolytemia	not done	not done	NR	mono	stable	adm	drugs	recurrance	recurrance
112	1013647	2.6	2	4.4	2	26	2	11	55	12	12	40	13	CAD	CABG	Triple vessel disease	STEMI	poly	unstable	<24hr	E.cardioversion	recurrance	death
113	1016344	2.5	2	4	2	28	2	9	46	10	10	37	12	CAD	CABG	Triple vessel disease	STEMI	poly	unstable	<24hr	E.cardioversion	death	death

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Date 8/9/2021

The Principal,
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Subject: Approval of Plan of Thesis.

This is to inform you that the Faculty of Medical Sciences in its meeting held on 22.01.2021 has approved the plan of thesis of Dr Akshay Mahajan, a student of your college for DM Cardiology along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Clinical profile and outcome of patients with ventricular tachycardia

Supervisor
Dr. Naved Aslam
Prof.,
Dept. of Cardiology,
DMCH, Ludhiana.

Co-Supervisor
Dr. Bhupinder Singh
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The student has to submit the three copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*

CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH VENTRICULAR TACHYCARDIA

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

DOCTORATE OF MEDICINE

IN

CARDIOLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

MARCH, 2020

DR. AKSHAY MAHAJAN

DEPARTMENT OF CARDIOLOGY

DAYANAND MEDICAL COLLEGE & HOSPITAL

LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH VENTRICULAR TACHYCARDIA
For the degree of	D.M Cardiology
Name of the candidate	Dr. Akshay Mahajan
Supervisor	Dr. Naved Aslam, Professor, Department of Cardiology, DMCH, Ludhiana
Co-supervisor	Dr. Bhupinder Singh, Associate Professor, Department of Cardiology, DMCH, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Ventricular tachycardia (VT) represents the most frequent cause of sudden cardiac death. Information on clinical characteristics, acute management and outcome of patients with sustained VT is limited in our part of world.

Aim of Study: The aim of this study was to analyze the demography, hemodynamics, ECG features, underlying disease, mode of termination and outcome of patients presenting with VT.

Materials & Methods: All cases with definite ventricular tachycardia already admitted in the hospital or presenting in emergency department would be included in the study.

Clinical Significance: The study of etiology and prognosis of patients with VT would help in effective management of patients with VT and possibly identify predictors of future arrhythmic events.

Keywords: Ventricular tachycardia, Etiology, Cardiomyopathy, Hemodynamics.

(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-Supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH VENTRICULAR TACHYCARDIA"** exist in the Department of Cardiology, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate himself/herself under our supervision and guidance.

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Date : 12/05/20

APPROVAL PROFORMA
BY RESEARCH & ETHICAL COMMITTEE
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Name of candidate	Dr. Akshay Mahajan
Department	Cardiology
Topic of Thesis	CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH VENTRICULAR TACHYCARDIA
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Date of enrollment	10.09.2019
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Signature of Members of Research Committee with Stamp	**Signature of Members of Ethics Committee with Stamp**
Dr. Jaspal Singh (Professor & Head, Surgery)	Mr. A K Jindal (Member, Ethics Committee)
Dr. Dinesh Gupta (Professor & Head, Medicine)	Dr. Anu Sharma (Professor, Anatomy)
Dr. Sandeep Kaushal (Assistant Dean Academics)	Dr. Deepinder K Chhina (Professor & Head, Microbiology)
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PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

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3	COURSE OF STUDY AND SUBJECT	DM CARDIOLOGY
4	DATE OF ADMISSION OF COURSE	10.09.2019
5	TITLE OF THE TOPIC	CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH VENTRICULAR TACHYCARDIA

6 BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Ventricular tachycardia is a common arrhythmia in patients with structural heart disease and heart failure, and is now seen more frequently as these patients survive longer with modern therapies. Ventricular tachycardia (VT) is defined as a series of three or more ventricular complexes occurring at a rate of 100 to 250 beats per minute. It is the most frequently encountered life threatening arrhythmia. Ventricular tachycardia (VT) can be classified according to duration, morphology, and hemodynamic effect. Nonsustained VT terminates spontaneously without substantial hemodynamic compromise. Sustained VT is VT that lasts longer than 30 seconds and/or requires an intervention for termination or produces severe hemodynamic compromise or syncope before terminating spontaneously.[1]

Etiology of ventricular tachycardia is an important consideration in treatment and prognosis of ventricular tachycardia. Ventricular tachycardia accounts for approximately 8% of cases of wide complex tachycardia.[2] The most common cause of ventricular tachycardia is underlying ischemic heart disease. Other causes include adult and congenital structural heart disease, acquired and inherited channelopathies, infiltrative cardiomyopathy, electrolyte imbalances (hypokalemia, hypocalcemia, hypomagnesemia), illicit drugs such as cocaine or methamphetamine, and digitalis toxicity.[3] Infiltrative cardiomyopathy can result from: systemic lupus erythematosus, sarcoidosis, amyloidosis, rheumatoid arthritis, and hemochromatosis.[4] A common trigger is hypokalemia followed by hypomagnesemia. Some cases may also be triggered by hyperkalemia, especially in patients with structural heart disease. Sleep apnea and atrial fibrillation can also trigger. Whereas in young people, the cause of VT includes Myocarditis, Hypertrophic cardiomyopathy, Long QT syndrome, Right ventricular cardiomyopathy and congenital coronary artery anomalies. The pathogenesis of VT depends upon its aetiology and type. In monomorphic VT the impulse is either being generated from increased

automaticity of a single point in either left or right ventricle, or due to a re-entry circuit in the ventricle. From clinical studies as well as animal models of VT in association with myocardial infarction (MI), it can be reasonably assumed that the underlying mechanism is re- entry. Re-entry is the mechanism underlying the VT associated with healed or healing MI in more than ninety five percent of cases. The most common cause of monomorphic VT is myocardial scarring from a previous MI. The scar cannot conduct electricity so there is a potential circuit around the scar that results in tachycardia. In case of idiopathic dilated cardiomyopathy, more than one third of the patients have sustained Bundle Branch Re- entry (BBR) as the cause of monomorphic VT.[5] Incidence of ventricular arrhythmia is 51.9 per 1,00,000. Idiopathic VT accounts for 10% of VT cases. Incidence of ventricular tachycardia increase with age. Women have a higher incidence of symptomatic PVC. The prevalence of VT is 16% in men and 15% in women with CAD, (% in men and 8% in women with hypertension, valvular disease or cardiomyopathy without CAD, and 3% in men and 2% in women with no cardiovascular disease.[6] Ventricular arrhythmias can present with wide range of symptoms such as palpitations, shortness of breath, chest pain, dizziness, syncope. Patients presenting with ventricular tachycardia are more likely to be older with a history of heart disease. A past medical history of heart failure, recent angina, and prior myocardial infarction all have a greater than 9% positive predictive value for ventricular tachycardia.[7] Precipitating factors such as exertional or emotional stress, concurrent medications or illness. Hypotension during VT is common and the clinical significance of hemodynamic status is important for the management of VT. The risk of sudden cardiac death in VT is related to the underlying heart disease as evidenced by increased incidence of sudden cardiac death in coronary artery particularly in patients having poor left ventricular function.

This prospective cohort study is aimed at studying patients presenting with sustained VT in our hospital to analyze the demographic and

hemodynamic features, ECG characteristics, underlying disease, mode of termination and clinical outcome following discharge.

6.2 REVIEW OF LITERATURE

Ventricular tachycardia is one of the most important cause of broad complex tachycardia (BCT) accounting for 80% of all BCT.[2] Ventricular tahycardia is defined as "as an arrhythmia of ≥3 consecutive complexes originating in the ventricles at a rate of >100 bpm.

Types of Ventricular tachycardia:

1. Sustained VT: >30s or requiring termination due to hemodynamic compromise in <30s.
2. Non sustained/ unsustained VT: ≥3beats, terminating spontaneously.
3. Monomorphic VT: Stable single QRS morphology from beat to beat.
4. Polymorphic VT: Changing or multiform QRS morphology from beat to beat.
5. Bidirectional VT: VT with a beat to beat alteration in QRS frontal plane axis, often seen in the setting of digitalis toxicity or catecholaminergic polymorphic VT.

Risk factors for ventricular tachycardia include prior history of hypertension, prior MI, ST-segment changes at presentation, and chronic obstructive pulmonary disease. The most important etiological factors of VT include:

1. Ischemic heart disease
2. Strucural heart disease
3. Inherent and acquired channelopathies
4. Electrolyte imbalance
5. Sympathomimetic agents
6. Digitalis toxicity
7. Systemic diseases causing infiltrative cardiomyopathy
8. Idiopathic

Ventricular tachycardia can be either asymptomatic or can be associated with symptoms such as palpitations, syncope or chest pain. Signs of ventricular tachycardia include hypotension, signs of diminished perfusion, raised JVP, variation in intensity of first heart sound.

Akhtar (1990) [8] studied the spectrum of VT and observed that CAD is the most common cause of sustained monomorphic VT. Polymorphic VT is caused by both acquired and congenital forms of QT prolongation. The acquired form of QT prolongation is seen in association with the use of antiarrhythmic drugs and in the presence of hypokalemia and hypomagnesemia. Most but not all the patients with polymorphic VT in the absence of QT prolongation have CAD.

Domanovits H, Paulis M, Nikfard Jam M et al (1999) [9] studied sustained VT in 75 patients in the emergency department and observed that ischaemic heart disease was underlying disorder in majority of the patients (76%). Hemodynamic instability was present in 23 % patients. Polymorphic VT was present in 9% of patients. Non sustained VT was present in 4% patients. Mortality was 7%. Majority of the patients (88%) were initially treated with antiarrhythmic medication, but half of these patients needed DC cardioversion finally for the definitive termination of VT. Thus 50% of patients needed both medical management and DC shock for management of VT.

Elliot PM, Sharma S, Varnava A (1999) [10] studied survival after sustained VT in patients with hypertrophic cardiomyopathy and found that the patients with hypertrophic cardiomyopathy who survive an episode of VT remain at risk for recurrent event. Intracardiac defibrillator (ICD) therapy appears to offer the best potential benefit regarding outcome.

Steinberg JS, Gaur A et al (1999) [11] studied new onset sustained VT after cardiac surgery and found that the first presentation of sustained monomorphic VT in the recovery period after coronary artery bypass grafting (CABG) is uncommon, but the incidence is high in specific clinical subsets. Placement of a bypass graft across a non-collateralized total occlusion vessel supplying an infarct zone was strongly and independently associated with the development of VT.

Kautzner J (2000) [12] studied clinical spectrum of VT and observed that among the monomorphic VT, the sustained VT after myocardial infarction is the most frequent form, accounting for more than 70% of all VT cases.

Pires LA, Lehmann MH, Buxton AE et al (2001) [13] studied inducibility and prognosis of in-hospital versus out of hospital identified non sustained VT (NSVT) in patients with CAD and found that in patients with CAD and impaired left ventricular ejection fraction (LVEF), asymptomatic NSVT identified in hospital, compared with that identified out of hospital is associated with higher rate of induction of sustained VT and overall mortality , therefore in similar patients, the clinical setting in which non sustained VT is discovered should be taken in to consideration while formulating patient risk and treatment.

Nakagawa M, Takashashi N, Nobe S et al (2002) [14] studied gender differences in various types of VT and observed that gender specific differences exist in incidence and age distribution of various types of idiopathic VT.

Di Lenarda A, Pinamonti B, Mestroni L et al (2004) [15] studied the natural history of dilated cardiomyopathy (DCM) in 581 patients and observed that long term optimised treatment with ACE inhibitors (in 90% of cases) and beta blockers (in 87% of cases) was associated with a remarkable clinical improvement in 50% of the patients and apparent healing in 16% of the cases. The risk of sudden cardiac death (SCD) was increased particularly in the patients with long term persistant or progressive left ventricular dilation and dysfunction.

Cuoco FA, Singh SN (2005) [16] studied NSVT in dilated cardiomyopathy (DCM) and observed that NSVT is common in patients with DCM of both ischaemic and non ischaemic origin. Recent studies suggest that NSVT may be a marker but not a significant predictor of mortality and sudden cardiac death (SCD).

Harris KC, Potts JE et al (2006) [17] studied right ventricular outflow tract tachycardia (RVOT) in children and observed that clinical spectrum and management of RVOT in children are diverse. Both medical therapy and observation alone were associated with a reduction in ectopy.

Varriale P, Leonardi M (2006) [18] studied polymorphic VT in the coronary care unit and observed that acute myocardial infarction is the most common cause of polymorphic VT associated with a normal QT

interval. Polymorphic VT with or without QT prolongation is a sporadic tachyarrhythmia that has frequent malignant potential for cardiac arrest. Successful management mandates emergency defibrillation for cardiac arrest and other appropriate measures for suppression of polymorphic VT. The prognosis of polymorphic VT is improved when the cause is correctly identified and promptly treated.

Kanjwal K, Imraan N, Grubb B (2008) [19] studied the troponin elevation in patients with various tachycardias and normal epicardial coronaries and observed that the patients with elevated troponin due to conditions other than acute coronary syndrome can receive inappropriate and delayed definitive diagnosis and treatment. So, in the patients admitted within the frame work of tachyarrhythmia without hemodynamic instability, the decision whether to obtain serum markers of myocardial ischaemia should cover a comprehensive clinical context.

Suesaowalak M, Khongphattanayothiu A et al (2008) [20] studied idiopathic left ventricular tachycardia in children and found that median age at presentation was 9.5 years. Majority of the patients responded to intravenous verapamil.

Aleksova, Aneta, Sabbadine, Gustone et al (2009) [21] studied the frequency with which the patients effected by the dilated cardiomyopathy (DCM) are diagnosed in the asymptomatic state and factors influencing their outcome. They observed that the proportion of asymptomatic patients with DCM at the moment of first evaluation is 30%. Among these, the patients without a previous history of heart failure had a less advanced disease and a trend for better long term quality of life and even better survival.

Patrizo Pascale, Jurg Schlapter, Mauro Oddo et al (2009) [22] studied VT in CAD and observed that inferior scars may be more prone to develop VT as compared to anterior scars.

K Kjeldsen (2010) [23] studied hypokalemia and sudden cardiac death (SCD) and observed that hypokalemia is present in 7 % to 17% of the patients with cardiovascular disease. Up to 40% of the patients on diuretics suffer from hypokalemia. Inadequate management of

hypokalemia was found in 24% of hospitalized patients. Hypokalemia is associated with ihcreased risk of cardiac arrhythmia in patients with cardiovascular disease as well as increased all-cause mortality by up to ten fold.

Mazik S, Lazovic B, Delic M et al (2012) [24] worked on the arrhythmogenic right ventricular dysplasia (ARVD) as a cause of sudden cardiac death (SCD) in the young people and found that the incidence of ARVD is about 1/1000 to 1/5000 and the presentation is usually related to VT, syncope, presyncope or ventricular fibrillation (VF) leading to cardiac arrest. It occurs mostly in young people and athletes. The intracardiac defibrillator (ICD) is the most effective safeguard against sudden cardiac death (SCD).

Mehta RH, Yu J, Piccini JP et al (2012) [25] studied the prognostic significance of post procedure sustained VT in patients undergoing primary percutaneous coronary intervention (PPCI) for ST elevation MI (STEMI) and found that out of 3485 patients undergoing PPCI in whom VT/VF did not develop before or during procedure, 5.2% developed VT/VF after PPCI. Most VT/VF occurred in the first forty eight hours after procedure. Majority of these patients were killip class >1 on presentation.

Nerry PB, Leung E et al (2012) [26] studied arrhythmias in patients of sarcoidosis and proposed that sarcoidosis should be considered in patients aged less than fifty five years presenting with unexplained AV block and in patients with sustained VT.

Aysen Agir, Serdar Bozyd, Umut Celikyurt et al (2014) [27] studied ARVD in pregnancy and observed that pregnancy is well tolerated in women affected by ARVD and can be managed successfully with close monitoring and anti-arrhythmic drugs when necessary. ICD implantation should also be considered to prevent sudden cardiac death (SCD) during pregnancy to prevent life of mother. Pregnancy seems not to cause an increase in major ICD related complications.

6.3 AIMS AND OBJECTIVES OF THE STUDY

- To study the demographical profile of Ventricular Tachycardia patients.
- To study the clinical profile of Ventricular Tachycardia patients.
- To evaluate the hemodynamic, underlying disease, tachycardia termination and outcome of patients with ventricular tachycardia.

7.0 MATERIALS AND METHODS

7.1 SOURCE OF DATA:

All the patients admitted in the cardiology department of DMC hospital with diagnosis of VT on 12-lead ECG or non invasive monitor.

7.2 METHOD OF COLLECTION OF DATA

Inclusion Criteria: All cases with definite ventricular tachycardia already admitted in the hospital or presenting in emergency department would be included in the study.

Exclusion Criteria:

1. Patients without known etiology who died early after hospitalization thereby precluding further evaluation.
2. Patients refusal for further evaluation.
3. Patients with recurrent episodes of VT, only the first episode was considered as index event and evaluated.
4. Patients with out of hospital pulse less VT that was terminated before arrival to our hospital but had no documentation of the same.
5. Patients with pre-implanted implantable cardioverter defibrillator (ICD) because of the different management of such group.

Study protocol: Each patient will be placed on non invasive monitor for heart rate and rhythm, blood pressure and pulse oximetery.

ECG interpretation: Standard 12 lead ECG during and after VT will be recorded in all patients. The ECG diagnosis of VT will be made by applying "The Brugada algorithm" 7 and/or "The aVR 'Vereckei' algorithm.

The ventricular rate, QRS width, VT morphology will be noted. Ventricular tachycardia will be defined as ≥3 sequential wide complex beats arising from ventricles, rate ≥100 beats per minute.

These patients will be divided into two sub-groups: hemodynamically stable and hemodynamically unstable. Hemodynamic instability is defined as need for immediate intervention due to loss of consciousness, hypotension or occurrence of congestive

heart failure. Otherwise patients will be defined as hemodynamically stable.

Other relevant investigations:

- Standard PA chest radiograph
- Serum electrolytes: Potassium, Magnesium, Calcium.
- Echocardiography: To evaluate Ejection fraction, Left ventricular dysfunction

VT termination and follow-up

The therapy for termination of VT protocol will be as per latest ACLS guidelines. Patients who are hemodynamically unstable will be administered electrical cardioversion. 100J of synchronised direct current (DC) will be given under proper sedation and analgesia. The energy will be subsequently increased to a maximum of 200J wherever required. Pulse less VT patients will be administered cardiopulmonary resuscitation (CPR as per ACLS guidelines. Intravenous antiarryhmics drugs in recommended doses will be given to hemodynamically stable patients. The choice of antiarrhythmic drug will be based on attending physician's discretion. Electrical cardioversion will be given to those patients who became secondarily unstable or in whom antiarrhythmic drugs failed to terminate VT. Follow-up information of each patient regarding hospital discharge or death will be obtained.

Study design: Prospective observational study.

Study place: Department of cardiology, DMCH, Ludhiana.

Study period: July 2020 to March 2021.

Ethical clearance will be sought from Institutional Ethics Committee of DMCH, Ludhiana prior to enrollment.

Written and informed consent will be obtained from eligible patients prior to enrollment into study.

Endpoints:

Outcome:

Primary outcome: VT recurrence and cardiovascular death.

Secondary outcome: Hospitalization and cardiovascular death.

Statistical analysis: Data will be described in terms of range; mean ±standard deviation (± SD), median, frequencies (number of cases) and relative frequencies (percentages) as appropriate. For comparing categorical data, Chi square ($\chi 2$) test will be performed and exact test was used when the expected frequency is less than 5. A probability value (p value) less than 0.05 will be considered statistically significant. All statistical calculations will be done using SPSS (Statistical Package for the Social Science) SPSS 21 version statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

NO

7.4 HAS ETHICAL CLEARENCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

ATTACHED HEREWITH

8.0 LIST OF REFERENCES

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PROFORMA

Name:

Age:

Sex:

MRD. No.

Address:

Mobile No.:

Chief complaints:

Chest pain

Shortness of breath

Palpitations

Syncope

Presyncope

Dizziness

Fatigue

NYHA (I, II, III, IV)

Diagnosis-

Past History

Hypertension :

Bronchial Asthma:

Tuberculosis:

CVA:

Thyroid Disoders:

Signature of Candidate

Signature of Supervisor

DIABETES MELLITUS:

Any surgical h/o:

Personal History

Smoker/Non Smoker:

Alcoholic/Non-Alcoholic:

Tobacco addict:

Family History

Diabetes Mellitus:

Hypertension:

CVA:

Prior drugs history-

-Amiodarone

-Cardiac glycosides

-Mexilitine

-Beta blocker

-Calcium channel blocker

Examination

GPE

Anemia :

Cyanosis :

Jaundice :

Oedema :

Pulse :

Blood Pressure :

Signature of Candidate

Signature of Supervisor

Respiratory Rate:

Temperature :

System Examination

-Abdomen

-CVS:

-Chest:

-CNS:

Investigations-

Lab parameters-

-Hb

-Urea

-Creatinine

-Sodium

- Potassium

-Calcium

-Magnesium

-Albumin

-SGPT

-SGOT

-Trop I

-BNP

-CPK MB

Signature of Candidate

Signature of Supervisor

ECG

- Ventricular rate
- AV Dissociation
- QRS width
- VT morphology
- QTc

Echocardiography Parameters-

-LVEF

-IVST(D) (S)

-LVID(D) (S)

-LVPW(D) (S)

-Grade of diastolic dysfunction

-≥Mild MR

Causes of VT-

-IHD

-Non ischemic cardiomyopathy

-Diselectrolytemia

- Others

CAG-

-SVD

_DVD

-TVD

-Coronary slow flow

- Normal

Signature of Candidate

Signature of Supervisor

Cardiac MRI-

Hemodynamically

-Stable

-Unstable

Morphology-

-Monomorphic

-Polymorphic

-Bidirectional

Pattern of vt-

-Sustained

-Non sustained

VT termination-

-Spontaneous

-Electrical Cardioversion

-Drugs (amiodarone, lignocaine,others)

Drugs at discharge-

-Amiodarone

-Cardiac glycosides

-Mexilitine

-Beta blocker

-Calcium channel blocker

In Hospital Outcome-

-No Recurrence

Signature of Candidate

Signature of Supervisor

-Recurrence

-Mortality

6 month Outcome-

-Recurrence

-Mortality

-Major adverse cardiac events

Signature of Candidate

Signature of Supervisor

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o.....
.....am willing to participate in the study **"CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH VENTRICULAR TACHYCARDIA"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

.....
Signature of the Witness

Name:

.....
Signature of the patient

Signature of Candidate

Signature of Supervisor

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य **"CLINICAL PROFILE AND OUTCOME OF PATIENTS WITH VENTRICULAR TACHYCARDIA"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____

गवाह के हस्ताक्षर

नाम:

_____

रोगी के हस्ताक्षर/अंगूठे का निशान

नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर